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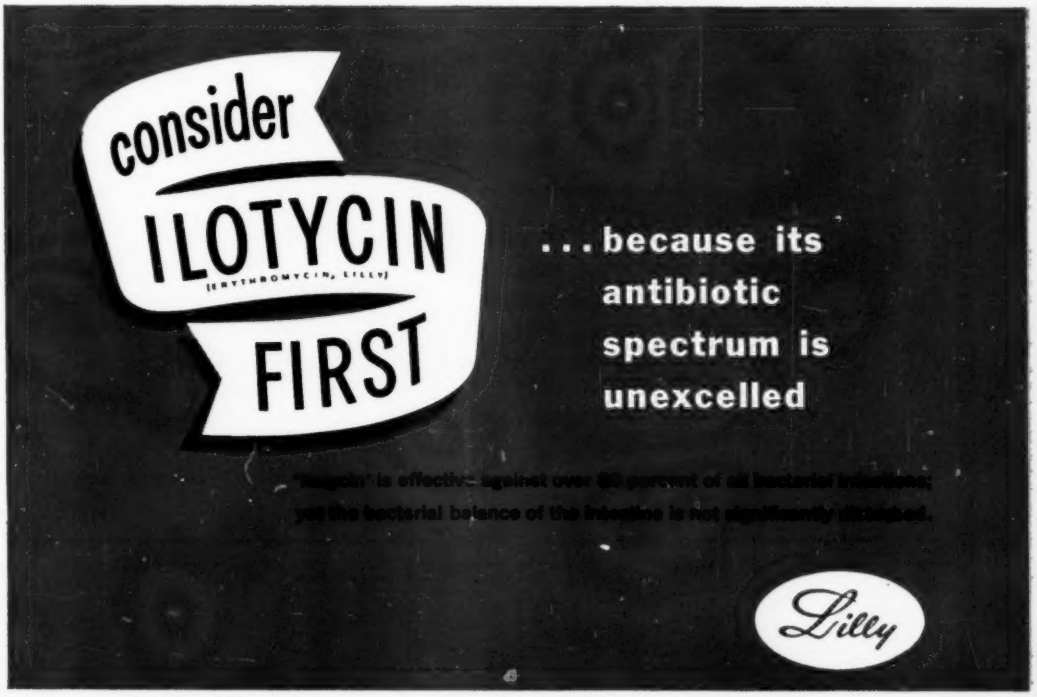
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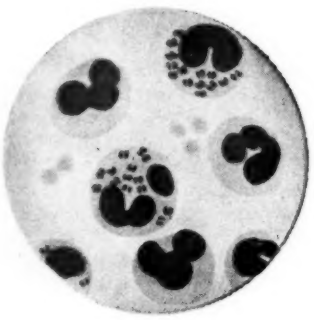

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(1) Yow, E. M.; Taylor, F. M.; Hirsch, J.; Frankel, R. A., & Carnes, H. E.: *J. Pediat.* **42**:151, 1953. (2) Dodd, K.: *J. Arkansas M. Soc.* **10**:174, 1954. (3) Hanbery, J. W.: *Neurology* **4**:301, 1954. (4) Miller, G.; Hansen, J. E., & Pollock, B. E.: *Am. Heart J.* **47**:453, 1954. (5) Keefer, C. S., in Smith, A., & Wermer, P. L.: Modern Treatment, New York, Paul B. Hoeber, Inc., 1953, p. 65.

Minnesota Medicine

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MANAGEMENT OF MASSIVE HEMATEMESIS OF UNDETERMINED CAUSE

CHARLES E. REA, M.D.
Saint Paul, Minnesota

THE PURPOSE of this paper is to review the treatment of our cases of massive hematemesis in which the source of bleeding was not known preoperatively. Also, an evaluation of blind gastrectomy in such cases will be made.

Given a patient with massive hematemesis, the cause of the bleeding is usually due to either a duodenal ulcer, gastric ulcer, carcinoma of the stomach, gastric erosions, or esophageal varices. When any one of these lesions is found, the surgeon can institute definitive treatment. However, most surgeons have had the experience of operating upon patients in whom hematemesis could not be controlled by conservative means, and in whom no obvious cause for the bleeding was found. This occurs in as high as one-quarter or one-third of the patients operated upon for hematemesis so the problem is of more than academic interest.

When a surgeon operates upon a patient who has had a massive hematemesis, and the source of bleeding is not known preoperatively, he usually opens the stomach and duodenum to inspect the interior for the bleeding source. Palpation of the stomach and duodenum without opening these organs is a rather unsatisfactory procedure. If a duodenal ulcer is found, but is not actively bleeding, a search should be made of the stomach to be sure there are no bleeding lesions there. We routinely open the stomach and duodenum in all such cases because, on two occasions while a chronic duodenal ulcer was found, the bleeding was from an unsuspected gastric ulcer.

If, on inspecting the stomach and the first portion of the duodenum, the source of bleeding is still not found, a careful search of the upper end of the stomach, especially around the esophageal opening, and also the entire duodenum should be made. Gilchrist and Chun report a series[2] of eleven cases. In three instances fatal bleeding occurred from lesions 3.5 to 7 centimeters from the esophagus. In our experience, we have had a hemangioma in the second portion of the duodenum and also a leomyosarcoma near the ligament of Treitz. Neither of these lesions was discovered until autopsy but the futility of doing the conventional gastric resection with removal of 75 per cent of the stomach in such cases is apparent.

If, after careful examination of the entire stomach and duodenum no lesion is found, many surgeons perform a gastric resection, removing about 75 per cent of the stomach. This was popularized by Wangensteen[3] in 1945. More recently Cooper and Ferguson[1] have reported their experiences with this procedure. In their eighteen patients who received operations as an emergency measure for the control of upper gastro-intestinal hemorrhage, six showed no evidence of the source of bleeding even after thorough exploration. In each instance a resectable mucosal lesion or acute ulceration was established as the source of bleeding when the stomach was studied by the pathologist. In five of the patients, a subtotal gastric resection was done. In the one patient in whom a resection was not done the patient subsequently bled to death from a resectable superficial gastric erosion high on the lesser curvature of the stomach.

If one suspects gastric erosions, one should

Presented at the annual meeting of the Southern Minnesota Medical Association, Winona, Minnesota, September 13, 1954.

probably do an infradiaphragmatic vagotomy at the same time as the resection. It is interesting that of three patients with demonstrable bleeding gastric erosions that have come to our attention, one occurred after intensive hormone therapy for the menopause, the other after cortisone therapy for arthritis and the third in a woman five months pregnant. The hormonal factor may be of etiological significance.

In our series, there were two patients in whom multiple gastric erosions were found by the pathologist in the resected stomach. In both of these cases a three-quarter gastric resection had been done but the lesions were not diagnosed at the time of exploration.

If no lesion is found on examining the open stomach and duodenum to account for the massive hematemesis, why not simply close the incision without a resection? It appears that a gastric resection is justified in these instances from a review of our cases (Table I). Although in two cases gastric resection was of no value, in eight cases it was of definite value as the bleeding source was removed. In two cases it was of questionable value as no definite lesion was found, although the bleeding was controlled. If, after a blind gastric resection, the patient still bleeds, what should be done? Most surgeons would try conservative measures at first, but if these are of no avail, further resection of the stomach is usually resorted to. It should be emphasized that a careful search of the remaining stomach and the duodenum to the ligament of Treitz should be made because an obscure lesion, previously undiagnosed, may be found in these regions.

TABLE I.

No. of Cases	Lesion	Gastric Resection	Benefit	Comment
1	Hemangioma—2nd portion duodeum	+	0	Lesion found at autopsy
1	Leiomyosarcoma—3rd portion duodeum near ligament of Treitz	+	0	Lesion found at autopsy
2	Gastric erosions	+	+	Not recognized at operation
3	Duodenal ulcers	+	+	
2	Gastric ulcers	+	+	Associated with inactive chronic duodenal ulcer
1	Bleeding esophageal varices	+	+	Esophagogastrectomy performed
2	No definite lesion found	+	+	
12				

Summary

In most cases of massive hematemesis in which the source of bleeding is not known preoperatively, the cause will be definitely found at the time of operation. This occurred in eight of our twelve cases. However, in one-third of the cases a definite lesion was not found. In two of these four cases a gastric resection was performed and the cause of the bleeding was not found in the resected stomach. In the two other instances, lesions in the duodenum were found which were not amenable to gastric resection and in which a gastric resection would have been useless under any circumstance.

While our series is small, the following conclusions seem valid. A gastric resection is of value if the bleeding lesion is found in the stomach or duodenum (eight out of twelve of our cases). If the lesion is not found and a blind resection done, the surgeon may experience further difficulty in about half of the cases (two out of four of our cases).

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RECURRENT PEPTIC ULCER

GEORGE A. HALLENBECK, M.D.

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ALTHOUGH surgical treatment is ordinarily quite effective in eliminating the pathologic process and controlling the symptoms in properly chosen cases of gastric and duodenal ulcer, failures occur in some instances. One complication of such operations which usually requires further surgical treatment is recurrence of peptic ulceration. The present discussion is concerned with this problem.

Brief Résumé of the Pathologic Physiology of Peptic Ulceration

Peptic ulceration may develop in any part of man's gastrointestinal tract which can be bathed with acid peptic gastric juice: the stomach, the duodenum, the small intestine near a gastroenteric anastomosis, the ileum in the presence of a Meckel diverticulum containing ectopic gastric mucosa and occasionally near sites of ectopic gastric mucosa elsewhere. Continuous hypersecretion of highly acid gastric juice has been observed in patients with duodenal ulcer both during exacerbations of the disease and during its quiescent phase.[15] The duodenal content of patients with duodenal ulcer, both during fasting and after eating, tends to be more acid than does that of normal people.[3] However numerous other contributing factors may be, the facts remain that the presence of acid gastric juice is almost always necessary for the continued existence of benign peptic ulcers and that such ulcers almost invariably heal if the ulcerated area can be protected from the action of gastric juice by nonsurgical or surgical means.

Understanding of the problem has been greatly aided by experiments upon animals, too numerous to be presented in detail here, but which constitute a rewarding chapter in the field of experimental surgery. Peptic ulceration can be produced in dogs by surgical procedures, such as the Mann-Williamson operation,[16] which deprive the small intestine near a gastroenteric anastomosis of the neutralizing effect of bile and pancreatic juice or by methods, such as injection of histamine in beeswax,[11] which cause continuous hypersecretion of acid gastric juice. Results of many studies along these lines consistently support the hypothesis that peptic ulceration will occur in experimental animals when there is inadequate neutralization or excessive production of gastric juice. It is logical to apply these premises to man; indeed, they constitute the physiologic basis for present-day medical and surgical treatment of peptic ulcer. Recurrent ulceration, which follows surgical treatment for duodenal ulcer much more often than that for gastric ulcer, usually occurs in the jejunum adjacent to the site of gastrojejunostomy and can be looked upon as the result of failure of the original operation to reduce the rate of secretion of gastric juice below the level which can produce ulceration in the jejunum of a particular patient.

Prevention of Recurrence of Peptic Ulceration With Reference to Various Standard Operative Procedures

Gastroenterostomy.—The surgical procedure, gastroenterostomy, presents an entirely logical mechanical solution to the problem of pyloric obstruction and the operation has produced many brilliant results in this situation. During the first 3 decades of this century, it came to be used frequently for duodenal ulcer, with or without symptoms of obstruction, and was commonly followed by at least temporary healing of the ulcer and alleviation of symptoms. The physiologic basis for the improvement so often noted is not entirely clear. There is no reason to believe that gastroenterostomy reduces gastric secretion; in fact, experiments indicate that in the dog the operation can result in an increased secretion of gastric juice.[12,27]

Read at the annual meeting of the Northern Minnesota Medical Association, Willmar, Minnesota, September 10, 1954.

From the Section of Surgery, Mayo Clinic and Mayo Foundation.

The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Despite the many good results which followed gastroenterostomy when the operation was applied as a routine for patients with duodenal ulcers, the procedure fell into disrepute because of the distressingly high incidence of subsequent gastrojejunal ulceration. Data from a series of papers reviewed by Portis[20] indicate a reported incidence of gastrojejunal ulceration following gastroenterostomy varying between 1.4 per cent and 34 per cent. The first figure is far too low and a commonly quoted estimate of the incidence is 16 per cent. The interval between gastroenterostomy and a secondary surgical procedure for gastrojejunal ulcer may be long. In a study of 186 cases reported by Walters, Chance and Berkson,[24] this interval was 11.2 years. Thus the shorter the follow-up, the lower will the observed incidence probably be. Likewise, a lower incidence for recurrence can be expected if the operation is not used for younger people, especially men, with very pronounced ulcer diatheses, and a higher recurrence rate will follow inclusion of such patients in a series.

Both clinical experience and laboratory data provide information to help minimize occurrence of gastrojejunal ulceration after gastroenterostomy. The first warns the surgeon to avoid use of the operation in young and middle-aged people with very active ulcer symptoms and to consider its usefulness peculiar to a few older people whose symptoms are primarily due to cicatricial pyloric obstruction and whose physical status is so poor that more extensive operations would entail too great a risk. The second, based on the observation that, in dogs, establishment of a gastrojejunal stoma high in the stomach is followed by increased secretion from a Heidenhain pouch whereas establishment of a stoma in the antrum is not,[27] suggests that the stoma should be made low in the stomach. Ample laboratory data also indicate that anastomosis between proximal and distal loops of jejunum is to be avoided because it deviates neutralizing secretions from the stoma and promotes formation of gastrojejunal ulcers.[7]

Gastric Resection.—Use of gastric resection as treatment for duodenal ulcer grew from dissatisfaction with the high incidence of recurrence of ulcers following gastroenterostomy in most patients who require surgical treatment for the disease. Furthermore, the current practice of limiting recourse to surgery for patients with definite complications of duodenal ulcer has decreased the number of surgically treated patients with disease mild enough to permit expectation of good results with gastroenterostomy. Improvements in preoperative and postoperative care together with standardization of operative techniques, has lowered the risk of gastric resection to acceptable levels. The purpose of gastric resection is to reduce the total quantity of gastric juice secreted daily to levels which render subsequent ulceration at the gastroenteric stoma unlikely while still leaving enough stomach to permit eating with comfort and to allow maintenance of body weight.

Several techniques have been developed for reestablishment of gastrointestinal continuity after gastric resection. One would think that some type of gastroduodenostomy would most nearly restore normal anatomic relationships, render subsequent ulceration least likely and probably favor best digestion of food; this procedure has many partisans at present. Actually, the deformity of the duodenum often precludes satisfactory end-to-end gastroduodenostomy and the fixation of the duodenum commonly seen in patients with severe duodenal ulcer would often limit the extent of resection to unsafe levels if the operation were forced. Furthermore, the incidence of recurrent ulceration after the Billroth I procedure for duodenal ulcer has often been reported higher than that for resection with gastrojejunostomy. For these reasons, one of the variants of the Billroth II procedure in which the remaining stomach is joined to the jejunum is commonly used. Judging from the variations in preference shown by individual surgeons for postcolic or antecolic anastomoses, for the Hofmeister or "full" Polya operation and so forth, it seems fair to state that several procedures can give satisfactory results. There are, however, certain principles which must be followed if recurrent ulceration is to be kept at a minimum. These principles are:

1. *Removal of an adequate amount of stomach.* In the dog, 75 per cent of the stomach must be removed to prevent formation of ulcer in response to injections of histamine in beeswax, as Lannin,[14] in Wangensteen's laboratory, has

shown. In man, lower resections are followed by a higher incidence of recurrent ulceration than are higher resections. Figures of more than 9 per cent are common in series of patients from whom half or less of the stomach is removed,[22] values of around 4 to 5 per cent often accompany descriptions of removing two-thirds of the stomach[1] and recurrence rates of less than 1 per cent have been reported by surgeons who describe removing three-fourths of the stomach.[9,10] Because it has not seemed to me that increasing the extent of resection from two-thirds to three-fourths significantly increases the severity or incidence of postgastrectomy symptoms, I favor higher resections and try to remove three-fourths of the stomach.

2. *Complete removal of the pyloric (antral) mucosa.*—Experiments on dogs, particularly those of Dragstedt and his colleagues,[5,19] conclusively show that, under certain circumstances, the antrum may liberate a hormone which stimulates any remaining acid-secreting gastric mucosa to secrete large quantities of juice. Clinical experience amply proves that leaving some or all of the antrum in place is followed by an increased incidence of peptic ulceration.[13,17] One must conclude that though the duodenal ulcer itself can be safely left in place if this is technically advantageous, the antral mucosa must be totally removed if recurrence of ulcer is to be avoided.

3. *Use of as short a proximal loop of jejunum as is practicable.* Merendino and his associates,[18] in Wangensteen's laboratory, found that with resection of 75 per cent of the stomach of dogs, stomal ulcers developed in response to injections of histamine and beeswax if the proximal loop of jejunum was long but not if this portion was made short. The ability of jejunal contents distant from the duodenum to neutralize acid is less than that of contents obtained nearer the duodenum.[4] The corollary of this observation is that the afferent loop of jejunum should be made as short as practicable.

4. *Avoidance of any procedure such as entero-enterostomy which diverts duodenal contents away from the anastomosis.* This concept has been presented above with reference to gastroenterostomy.

If what has been recommended is followed carefully, it should be possible to keep the incidence of recurrent ulceration to no more than 3 per cent, and even lower figures have been reported.

Gastroenterostomy With Vagotomy.—At present, gastroenterostomy with vagotomy is enjoying extensive clinical trial. The purpose of adding vagotomy, of course, is to eliminate the cephalic phase of gastric secretion, stimulation of which is mediated by the vagus nerves. Though longer periods of observation will be needed to establish the incidence of recurrence of ulceration after this combined operation, present indications are that it lies between 5 and 8 per cent.[23] It is impossible to be certain how many of these recurrences are associated with incomplete vagotomy, but not all are.[2] If the pattern of recurrence of ulceration after the combined operation were to be similar to that after gastroenterostomy alone, the incidence of gastrojejunal ulcer would rise as the years pass. Vagotomy alone, as a primary procedure for duodenal ulcer, has been abandoned because of the high incidence of recurrences, failures of the stomach to empty properly, and diarrhea.

Other Procedures Currently Being Evaluated.—Though experience with certain other procedures is still insufficient to permit more than tentative conclusions, they deserve mention.

Hemigastrectomy and vagotomy are appealing because the procedure eliminates the cephalic and antral mechanisms for stimulating gastric secretion while leaving the reservoir function of the stomach less impaired than is the case with subtotal gastric resection. In a preliminary report, Farmer and Smithwick[8] noted that achlorhydria followed this procedure more regularly than was the case with either gastroenterostomy and vagotomy or with subtotal gastric resection. It is too early to determine the incidence of recurrence which will follow this operation. The procedure is based upon sound physiologic principles and deserves careful trial.

Subtotal gastrectomy with vagotomy has been appraised by Druckerman and his associates,[6] who reported no recurrent ulcers in a group of 165 patients who had undergone this operation, as compared with twelve recurrent ulcers (5.5 per

cent) in a group of 220 patients who had undergone partial gastrectomy alone. Morbidity, largely postoperative diarrhea, was greater in the group which had undergone gastric resection with vagotomy.

Wangensteen[25,26] has recently been trying two operations which have as their principle the removal of sufficient acid-secreting gastric mucosa that even though the antrum, with its potentiality of stimulating secretion of acid gastric juice, is preserved, so little acid juice can be secreted that ulcerogenesis is prevented. The first of these is segmental gastric resection, which consists of sleeve resection of the body of the stomach with preservation of the pyloric antrum and of a rim of stomach near the esophagus constituting only 5 to 10 per cent of the organ. The remaining upper remnant of stomach and the antrum are anastomosed and pyloroplasty is carried out to insure satisfactory gastric emptying. The procedure protects dogs against histamine-induced ulcers and, in 1953, Wangensteen had noted no recurrent ulcers in a group of ninety patients, some of whom had undergone the operation as early as 1949. The second of the procedures which Wangensteen[26] has been trying is termed "tubular gastric resection with transverse gastroplasty." In this operation a large portion of the acid-secreting gastric mucosa is removed in one piece, consisting of the anterior and posterior gastric walls and the intervening greater curvature of the stomach. The lesser curvature is left intact and vagus nerve fibers passing down it are not cut. Transverse closure of the resulting defect, supplemented by pyloroplasty if obstruction is present, completes the procedure. Tubular resection protects dogs against formation of ulcers when histamine in beeswax is given. It is too early to know what the incidence of recurrent ulcer may be after this procedure but Wangensteen[25] has reported none in a group of approximately ninety patients so treated over a period of a year and a half.

Diagnosis of Gastrojejunal Ulceration

Recurrent ulceration is less common after gastric resection for gastric ulcer than after the same operation done for duodenal ulcer. For example, Priestley and Gibson[21] reported the antecedent lesion to be gastric ulcer in only 1.4 per cent of 283 cases of gastrojejunal ulcer studied. The disease occurs predominantly in men. In a series of 301 patients with gastrojejunal ulcer studied by Walters, Chance and Berkson,[24] 91 per cent were men and 9 per cent were women.

In general, whenever gastroenterostomy or gastric resection has been performed for peptic ulcer and symptoms of ulcer again develop or hemorrhage from the stomach takes place, gastrojejunitis or gastrojejunal ulcer should be suspected. Roentgenologic demonstration of these lesions tends to be more difficult than is the case with primary gastric or duodenal ulcers. Thus, in one series of cases,[24] a positive roentgenologic diagnosis of gastrojejunal ulcer was made in only 50 per cent of cases of ulcer occurring after gastric resection and 37 per cent of those following gastroenterostomy. In some instances, gastroscopic examination may provide a positive diagnosis but a negative gastroscopic examination is not of significance because the ulcer may be hidden from view in the jejunum.

Gastrojejunocolic fistula, variously reported to occur in from about 6 to 15 per cent of patients with gastrojejunal ulcer, is demonstrated by roentgenologic examination of the colon with the aid of barium much more regularly than by similar examination of the stomach.

Hemorrhage occurs in approximately 50 per cent of cases and is not usually fatal.

In many instances, the diagnosis of gastrojejunal ulcer will be made only at the operating table and the patient's signs and symptoms of recurrence must often provide the indications for surgical operation in the absence of objective preoperative evidence for ulcer.

Treatment of Gastrojejunal Ulcer

Although trial of nonsurgical treatment for ulcer may be justified in some cases in which recurrent symptoms develop after operation for peptic ulcer, and continued nonsurgical treatment may be advisable in a few cases in which the patient's general condition precludes surgical operation at any but a prohibitive risk, the majority of patients are best treated surgically.

When gastrojejunal ulcer follows gastroenterostomy, the choice will lie between vagotomy alone, provided there is no evidence for obstruction, and gastric resection with or without vagotomy. Walters, Chance and Berkson[24] have com-

piled the most recent data from the Mayo Clinic in this regard and have reported that 78 per cent of twenty-seven patients obtained excellent results after vagotomy alone (no mortality) whereas 86.5 per cent of 111 patients obtained excellent results after the anastomosis made at gastroenterostomy had been disconnected and gastric resection performed (mortality rate 0.7 per cent). These data give gastric resection a slight edge over vagotomy for gastrojejunal ulcer following gastroenterostomy.

There are undoubtedly patients in whom the added security of vagotomy, performed in addition to gastric resection, compensates for the increased morbidity which the procedure is reported to impose and there remains room for the surgeon, giving consideration to the severity of the ulcer diathesis, to exercise his judgment in this regard. Simple disconnection of the gastroenteric anastomosis is mentioned only to condemn it, for results are usually poor.

In the presence of gastrojejunal ulcer following gastric resection, re-resection of the stomach is accompanied by a much higher operative mortality rate and gives poorer results than vagotomy alone. In the series of Walters, Chance and Berkson,[24] 70.5 per cent of seventy-eight patients who had gastrojejunal ulcers after gastric resection reported excellent results after vagotomy. The operative mortality rate was only 1.1 per cent. There was no difference in end results whether vagotomy was performed transthoracically or transabdominally.

Thus, data from the Mayo Clinic led to the conclusion that disconnection of the gastroenteric anastomosis, together with gastric resection, sometimes accompanied by vagotomy, is the procedure of choice for gastrojejunal ulcers following gastroenterostomy and that vagotomy alone is best for such ulcers following all but minimal gastric resections.

Patients with gastrocolic fistulas may be seen when in a state of good nutrition or may be encountered with moderate or severe degrees of malnutrition. Patients in poor condition become so not so much because of loss of foodstuffs into the colon as because of passage of feces into the stomach, with consequent gastroenteritis and anorexia. Because of this, establishment of a temporary colonic stoma proximal to the gastrocolic fistula has been proposed as a preliminary operation before definitive surgical treatment of the fistula and has been found effective in relieving the anorexia and improving nutrition. Though I would not hesitate to use this procedure if the general condition of a patient was poor enough that closure of the fistula and an appropriate operation for the ulcer diathesis would entail too much risk, experience at the clinic has indicated that most of the patients seen recently can be treated satisfactorily by a single operation. Thus, Walters, Chance and Berkson[24] have described twenty patients with gastrocolic fistulas treated between 1945 and 1952 with procedures of one stage. In sixteen of these cases the antecedent operation had been gastroenterostomy. In twelve of these, excision of the fistula was followed by gastric resection; in four by vagotomy. In four patients, the gastrocolic fistula followed gastric resection. Three of these were treated by gastric re-resection and vagotomy, one with excision of the fistula and vagotomy. There were no operative deaths. Of fifteen patients who could be followed, thirteen had excellent results.

Perspective on the Problem

This discussion, concerned as it is with the problem of recurrent ulceration after surgical treatment for peptic ulcer, should not lead to the erroneous conclusion that this combination is the only problem to be considered. The sacrifice of large amounts of stomach is followed by crippling postgastrectomy symptoms in a few patients and by less serious but very annoying symptoms, such as inability to eat more than small amounts at one time and inability to maintain acceptable body weight, in some others. Vagotomy is sometimes followed by delayed emptying of the stomach, epigastric bloating discomfort, vomiting and diarrhea; these may be only mildly annoying or in a few instances may be described as worse than the original disease. A low incidence of recurrent ulceration is only one, though a very important, criterion of a successful operation for peptic ulcer. It is fair to state that present-day surgical treatment for peptic ulcer is superior to that of even fifteen years ago with regard to mortality rate and incidence of undesirable by-products of operations, and that results compare favorably with those obtained from treatment of a host of other

surgically treated lesions. Yet the fact that some patients, perhaps 10 or 15 per cent of those operated upon, obtain results that fall short of reasonable satisfaction, properly prompts surgeons constantly to re-evaluate the procedures and to seek better means of treating this very common disease.

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STAPHYLOCOCCAL SEPSIS: CONTROL WITH ANTIBIOTICS

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THE problems encountered in the management of staphylococcal infections are increasing in their relative importance when compared to the problems in the control of other infections, particularly those caused by other Gram-positive bacteria. The treatment of severe staphylococcal infections, such as staphylococcal septicemia and acute endocarditis, is one of the most difficult problems in the management of infectious diseases.

The magnitude of the problem is better understood by considering that over 1000 strains of coagulase-positive staphylococci were isolated from 700 patients at the University of Minnesota Hospitals in a recent period of ten months. Most of these cultures were from localized infections. In the last two and one-half years we have studied approximately fifty patients with staphylococcal septicemia having a fatality rate of approximately 57 per cent. This high fatality rate is discouraging, since it has occurred under circumstances where many different antibiotics were available.

Staphylococci manifest considerable resistance to antibacterial agents which remain highly effective against other bacterial species. We have not encountered highly resistant strains of group A hemolytic streptococci, pneumococci, gonococci or the spirochete of syphilis. Strains of staphylococci which are now causing infections in hospitalized patients are so frequently resistant to penicillin, streptomycin and the tetracycline compounds (chlortetracycline, oxytetracycline and tetracycline), that a choice of the proper antibiotic for the treatment of severe staphylococcal sepsis becomes extremely difficult.

In order to control these highly fatal infections, certain helpful concepts may be formulated by considering the following pertinent points:

1. *Ubiquity of Staphylococci.*—The wide distribution of staphylococci and their presence on the skin results in frequent contamination of specimens which are submitted to the bacteriological laboratory. The presence of staphylococci in a blood culture must be evaluated as to whether the organisms represent contamination or the cause of an infection. This can be a very difficult problem. Helpful information is gained by the coagulase test, because if the culture coagulates plasma, it is probably a pathogenic strain. The coagulase test does not resolve the situation entirely, for coagulase-positive strains obtained from the skin of patients may present false information. This dilemma does not occur with other pathogens to the same degree that it does with staphylococci. Extreme precautions must be used in collecting and handling specimens. When obtaining blood for culture, the skin should be thoroughly cleaned with soap and water and dried with alcohol. Then it should be painted with tincture of iodine. The specimen is collected and the iodine can be removed with Richardson's solution. Attention to these precautions will contribute much to the proper evaluation of the patients' disease.

2. *Enzymatic and Toxic Properties of the Organism and Its Metabolic Products.*—The properties of the staphylococci are related to the manifestations of disease. During multiplication these micro-organisms produce potent toxins and enzymes which play major roles in the pathogenesis of the disease. Toxins arise which allow the bacteria to penetrate the tissue, cause necrosis, destroy leukocytes and fibrin, and hemolyze red blood cells. There is evidence that the pathogenic staphylococci are able to survive and multiply in the phagocytes. There is also a coagulating mechanism which probably is important in localizing the infections and causing formation of the abscesses. Some strains produce an enzyme, peni-

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TABLE I.
STAPHYLOCOCCUS AUREUS ISOLATED FROM INFECTIONS AT MINNEAPOLIS GENERAL HOSPITAL

	Resistant to >50 Units or Micrograms per ml.		
	1951	1952	1953
Penicillin	62.5%	57.2%	67.7%
Streptomycin	48.0	48.8	65.3
Oxytetracycline	38.0	47.5	62.7
Chlortetracycline	23.0	33.2	62.7
Chloramphenicol	25.0	2.5	0.65

TABLE II.
STAPHYLOCOCCUS AUREUS ISOLATED FROM INFECTIONS AT MINNEAPOLIS GENERAL HOSPITAL

	Resistant to >3.1 Units or Micrograms per ml.		
	1951	1952	1953
Bacitracin	10%	11.5%	7.8%
Neomycin	0	25.0	4.5
Erythromycin	—	0	21.0
Carbomycin	—	0	20.9

cillinase, which can quickly and completely inactivate penicillin.

3. *Tissue Reactions of the Invaded Host.*—The outstanding feature of staphylococcal infections is the formation of abscesses. Metastatic abscesses occur frequently and appear in the kidneys, liver, spleen, myocardium, lungs and brain with resultant complications of severe degree.[16] In the formation of an abscess, large numbers of polymorphonuclear leukocytes migrate to the site of bacterial localization, resulting in an inflammatory reaction with walling off of the infection. In the center there is a relatively avascular area with exudate, necrotic tissue, and large numbers of bacteria surrounded by the inflammatory wall. This is significant from the viewpoint of therapy. The amount of antibiotics which can diffuse into the center of the abscess is not known, yet this is the site where inhibition and destruction of the bacteria must be attained. Frequently a patient improves following therapy, treatment is discontinued, and relapse occurs. Awareness of the protection afforded the staphylococci in the abscess will be helpful in the management of this infection.

4. *Selection and Propagation of Strains Highly Resistant to Antibiotics.*—As mentioned, the problem of resistance to antibiotics is a challenging question. All strains have a measurable degree of resistance to antibiotics, but for practical application in therapy, arbitrary criteria for the delineation of degrees of resistance have been generally accepted. For this purpose strains of staphylococci are classified as follows: (a) sensitive, inhibited by 1.0 unit or microgram per ml. or less; (b) moderately resistant, inhibited by 1.0 to 10.0 units or micrograms per ml., and (c) resistant, inhibition by more than 10 units or micrograms of antibiotics per ml.

The incidence of penicillin-resistant strains in infections acquired outside of hospitals is less than in patients who acquire infections in hospitals. Approximately 25 per cent of staphylococcal infections isolated from out-patients show penicillin-resistant strains, and this figure has remained approximately the same for the last six years.[3,4,7,9,15,18] The incidence of penicillin-resistant strains causing infections in hospitalized patients has increased. At the time of introduction of penicillin in the treatment of infections, strains of resistant staphylococci were rare.[12,17] At the present time approximately 75 per cent of the staphylococci isolated from infections in hospitalized patients are resistant to the antibiotic.

A summary of the percentages of strains of staphylococci which were found to be resistant *in vitro* to over 50 units or micrograms of antibiotic per ml. for the years 1951 through 1953 is found in Table I. These strains were isolated from patients with staphylococcal infections at the Minneapolis General Hospital. Sixty-two and five-tenths per cent in 1951, 57.2 per cent in 1952 and 67.7 per cent in 1953 were not inhibited by 50 units of penicillin per ml.*

Results with streptomycin have been similar. A survey in 1949 revealed no strains of staphylococci to be resistant to this antibiotic. At Minneapolis General Hospital the percentage of strains which were resistant to 50 mcgm. per ml. increased from 48 per cent in 1951 to 65.3 per cent in 1953.

There has been increasing percentage of strains which are resistant to the tetracycline group of antibiotics. With oxytetracycline (terramycin), there was an increase from 38 per cent in 1951 to 47.5 per cent in 1952 to 62.7 per cent in 1953 of strains resistant to this high concentration. Results for chlortetracycline (aureomycin) are similar.

The data in regard to chloramphenicol (chloro-

*Acknowledgment is made of the technical services of Miss May Collin in performance of the sensitivity tests.

mycetin) are quite different from that of other antibiotics. There has been a decrease in the percentage of chloramphenicol-resistant strains since 1951. There was a reduction in the use of this antibiotic in 1951 and the percentage of strains which were resistant to 50 micrograms per ml. decreased from 25 per cent in 1951 to 2.5 per cent in 1952 and to 0.65 per cent in 1953. All strains of staphylococci which were obtained in 1953 were resistant to one microgram per ml. and 95 per cent were inhibited by 12.5 micrograms per ml.

A summary of the percentages of strains which are resistant to 3.1 mcgm. per ml. of other antibiotics is found in Table II. These strains were also isolated at the Minneapolis General Hospital from 1951 through 1953. Approximately 10 per cent of the strains were moderately resistant to bacitracin each year. Bacitracin was used topically in most cases, and employed parenterally only occasionally in the treatment of patients with staphylococcal septicemia. There has been little evidence of increasing resistance to neomycin, except in 1952, when 25 per cent of the strains exhibited moderate resistance. This was not true in 1953 when only 4.5 per cent were moderately resistant to neomycin.

There is evidence of staphylococci manifesting increased resistance to erythromycin. In 1952 all strains that were studied at the Minneapolis General Hospital were sensitive. In 1953, 21 per cent of all strains examined were resistant to 3.1 mcgm. per ml. The majority of these strains required 200 to 400 mcgm. per ml. for inhibition of growth. Carbomycin (magnamycin) has not been used except in a few patients, but every strain of staphylococcus which has demonstrated resistance to erythromycin in this study, has also been resistant to carbomycin in similar concentrations. This indicates a cross resistance between these two antibiotics.

These data indicate the increasing incidence of antibiotic-resistant strains of pathogenic staphylococci in hospitalized patients. Newer antibiotics are being discovered, but they too are losing effectiveness and the present large numbers of antibiotics adds to the perplexity of the problem of management.

An attempt to explain the mechanism of the appearance of resistant strains is presented in Figure 1. There can exist in a population of antibiotic-sensitive staphylococci a few cells which are naturally resistant. During therapy with antibiotics there is destruction of the susceptible cells and survival of these relatively few resistant cells.

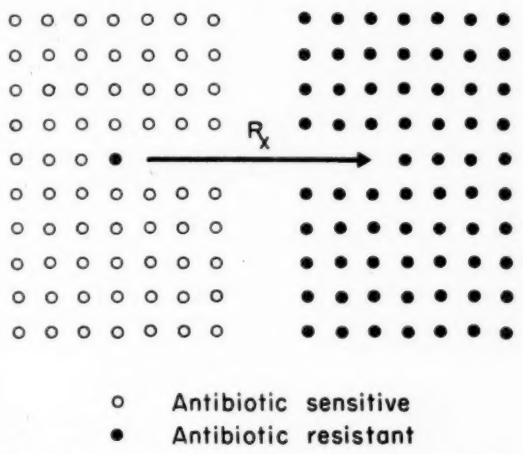

Fig. 1. Mechanism of the appearance of resistant strains of antibiotics.

The resistant organisms multiply rapidly and replace the antibiotic-sensitive staphylococci. This gives rise to an antibiotic-resistant culture of staphylococci.

5. *Epidemiology of Staphylococcal Infections.*—A knowledge of the mechanisms of spread of these bacteria is important, because if therapy of staphylococcal disease is so difficult, every precaution must be exercised in preventing such infections. The primary reservoir of staphylococci is the human nasopharynx. The carrier rate of pathogenic staphylococci in the general population is 25 to 50 per cent, whereas in hospital personnel the carrier rate is 50 to 75 per cent.[1,2,5,10,11,14] Nasal carriers are usually skin carriers of the same strains.[19] The spread of staphylococci from the nose to the intact and abraided skin occurs. The organisms are spread to bedclothes, dressings, dust and instruments.[6] Antibiotic-resistant strains find habitation in wounds and also in the noses and on the skin of hospital patients and personnel.[13] The cycle may be reduplicated many times in a hospital environment. To break this cycle great precautions must be taken in the care of indwelling venous catheters, surgical techniques, dressing of wounds

and burns, the instrumentation of the genitourinary tract and the treatment of staphylococcal infections of the skin.

The problem of antibiotic-resistant staphylococci appearing in a large hospital has been studied by Lepper et al[8] in the Municipal Contagious Disease Hospital of Chicago. Before introducing erythromycin in the wards, the carrier rate of erythromycin-resistant staphlococci in the hospital staff was determined. No strains were found that were resistant to this antibiotic. Erythromycin was then used for the treatment of many infections. After five months 75 per cent of the strains isolated from personnel required over 100 micrograms per ml. for inhibition. These results demonstrate the selection of strains of staphylococci which survive the action of antibiotics and consequently are resistant.

6. *Therapeutic Procedures.*—The concepts of therapy of severe staphylococcal infections must be based upon the factors which have been discussed. The most important point is to know when a staphylococcal infection is present. To begin therapy empirically with antibiotics before making an effort to obtain the etiologic agent is to invite trouble. A blood culture should be obtained from the febrile patient before therapy is started and changes can be made in management later when more information is obtained.

The abscess is a focus which may be impermeable to antibiotics. Bacteria may exist in the abscess for long periods of time, although high concentrations of antibiotics are present elsewhere in the body. The logical approach to this problem is the maintenance of high concentrations of properly selected antibiotics in the tissues for long periods of time, and surgical drainage when possible. This is in contrast to the treatment of infections caused by beta hemolytic streptococci, which usually produce an infiltrative type of infection rather than localized abscesses.

In some instances combined therapy is essential. The rationale of using at least two antibiotics in the treatment of severe staphylococcal disease is based on principles of genetic concepts of resistance. Since resistant cells are either present at the beginning of therapy or they arise by mutation during therapy, it is logical to assume that the chances of the resistant cell being resistant to two antibiotics are less than its chances of being resistant to a single antibiotic.

The antibiotics should be selected by an *in vitro* test of the susceptibility of the causative organisms to the antibiotic. The information gained by use of the tablet or disc method may be misleading in the case of staphylococci. The preferred technique for the staphylococci is the tube dilution method.

The management of the patient during the interval of waiting for results of cultures and sensitivity studies must necessarily be empirical. A presumptive etiological diagnosis of a bacterial infection and the most probable species of bacteria is made on the basis of history, physical examination and preliminary laboratory results. No rules can be made for the use of antibiotics. The tetracyclines or chloramphenicol are useful during this period, since they are active against many of the Gram-negative bacteria and some of the Gram-positive cocci. Concurrent use of one of these antibiotics with penicillin may be a satisfactory choice until bacteriological results are known.

Penicillin is the choice drug providing the causative organism displays *in vitro* sensitivity. Under these circumstances the following is suggested for stapylococcal septicemia: a total daily dose of at least 2 to 4 million units of crystalline penicillin G in aqueous solution is administered intramuscularly in divided doses every 4 hours. In addition, "booster doses" of 1 million units in 10 ml. of saline may be administered intravenously every twelve hours to produce high peak levels. The insoluble penicillin or deport penicillin should not be used in any staphylococcal infection for the prolonged low levels may allow the appearance of resistant forms. Therapy should be continued for a minimum of two weeks after the patient is afebrile. If the patient has normal renal function, bacitracin may be used simultaneously, but a total daily dose of 100,000 units in divided doses should not be exceeded and the urine should be examined daily for albuminuria. Bacitracin should be discontinued if nitrogen retention appears.

If the causative organism is resistant to penicillin and sensitive to erythromycin, then erythromycin should be given orally in total daily doses of 2 to 3 gm. or 500 mg. intravenously every 12 hours. Bacitracin can be administered concurrently with erythromycin.

Since there has been a decrease in the incidence of chloramphenicol-resistant strains of staphylo-

cocci in the last two years, this antibiotic may be valuable in the treatment of severe staphylococcal infections. In case the causative agent displays resistance to penicillin and to erythromycin, chloramphenical can be given orally in a total daily dose of 2 to 3 gm. or 500 mg. every eight hours intravenously. Bacitracin can be administered concurrently.

The other antibiotics, such as the tetracycline group, can be used when indicated as a result of sensitivity tests. Streptomycin is not desirable for long-term use because of the rapid appearance of resistant organisms. Neomycin can be used parenterally in a total daily dose of 1 gm. per day but should only be used as a last therapeutic resort, since irreversible deafness and renal damage can occur following the use of this antibiotic for periods necessary in the treatment of staphylococcal sepsis.

In addition, there should be good nursing care, adequate dietary intake, blood transfusions in anemic patients and surgical drainage of suppurative areas when possible.

Summary

In summary, generalized staphylococcal infections are severe and the fatality rate is high. Control and management of these infections should include measures of prevention. The toxic and enzymatic properties of the microorganisms, resistance to antibiotics, and the survival of organisms in abscesses are some of the problems to be surmounted. Treatment will be more successful when the identity of the causative agent and its sensitivity to antibiotics are known. Management should be immediate, aggressive and continued until all signs of infection have subsided.

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ACUTE POISONING IN CHILDREN

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THE ACCIDENTAL death of a child is the dramatic and tragic finale of someone's mistake. In no other form of accidental death does this fact appear more obvious than in that resulting from acute poisoning. And yet in the United States each year over 1,000 deaths are due to accidental poisoning in children. This is just the number of deaths recorded. It is unfortunate that there are no accurate statistics as to the frequency of poisonings, but it has been estimated that one per cent of all pediatric hospital admissions each year are for accidental poisonings. Even this does not give one a complete picture, for there are many cases of poisoning that are treated in the home, in the physician's office, and as out-patients in the hospitals. There are no statistics to show how many of these represent near catastrophies. Nor are there any figures available to show the number of sequelae of poisoning such as esophageal strictures, blindness, cerebral damage, permanent liver damage, etc. During the year of 1952 the Minnesota Department of Health reported eleven deaths due to poisoning in the one to fourteen years age group. During the years 1951-1952 a private hospital in Minneapolis treated 168 cases of poisoning in children under the age of five years.

It is a well-known fact that causes of death in children have changed markedly in the last twenty years. In 1930 the mortality tables showed pneumonia and communicable diseases to rank first and second, respectively, as causes of death in the one- to four-year age group. Accidents ranked third. With the advent of the sulfonamides, and later the antibiotics, deaths from infectious diseases dropped. In 1954, accidents are the leading killers of our children.

The causes of fatal injuries are illustrated in Figure 1. Although poisonings represent what apparently is a small percentage of deaths, it is well to remember that the incidence of poisoning in children is inadequately represented by mortality tables. Statistics comparable to those illustrated were obtained by the Minnesota Department of Health for the year 1952.

The incidence of poisoning is greatest in the one-year to four-year range. Its peak is in the two-year to three-year age group (Fig. 2). Recalling the development of the child, it is during this period that the motor development is very rapid, enabling the child to walk, climb, pull open drawers, doors and cabinets. A child of this age is an explorer, an adventurer, and a taster. If he can pick it up, he will smell it and/or put it in his mouth.

The manner in which a child may become poisoned may be one of several. It may be by (a) inhalation, such as carbon monoxide or carbon tetrachloride; (b) by absorption through the skin as in the analine dye, mercury, or boric acid; (c) by injection as in errors in dosage of narcotics or sedatives or (d) by ingestion. The latter accounts for the majority of accidental poisonings in children. It may be the inherent nature of a child to put things into its mouth, but parents, and yea, we physicians, encourage this practice. Parents, in an attempt to administer medications to their children resort to various antics and systems of reward to achieve this end. We as physicians have condoned the practice of the pharmaceutical houses in their attempt to disguise their medications to have "eye appeal" and to "taste good like candy."

Symptomatology

The symptoms of acute poisoning will vary with the poison consumed, but in general will fall into one or more of the following groups.

1. Gastro-intestinal. Nausea, vomiting, and abdominal pain are most common symptoms encountered.

2. Respiratory. This may range from mild hyperpnea as seen in salicylate intoxication to complete respiratory collapse with loss of con-

sciousness as found in barbiturate or nicotine poisoning.

3. Circulatory. This may be manifested by cyanosis, hemorrhagic manifestations, and shock.

foregoing symptoms, and in whom adequate evidence for their cause cannot be determined, acute poisoning should be considered. The absence of a history of poisoning does not rule out its possi-

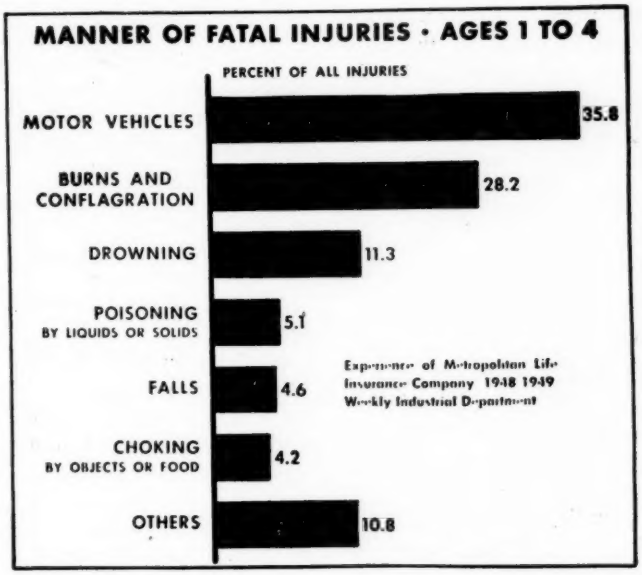

Fig. 1. Types of fatal injuries in the one-year to four-year age group.

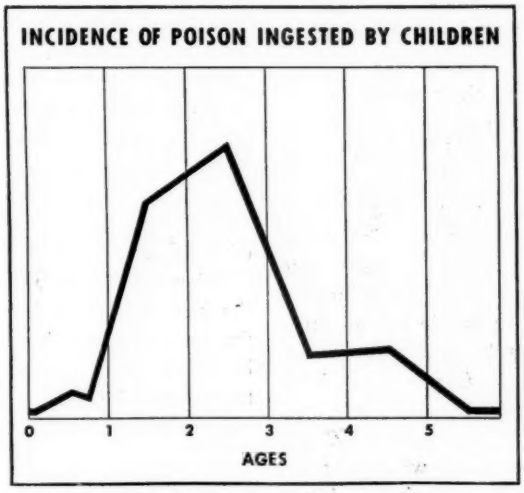

Fig. 2. Incidence of poisonings according to age.

4. Corrosive action. Evidence of erosion of oral and pharyngeal mucosa will occur with the injection of lye, phenol, alkalies and acids.

In any child who presents one or more of the

bility. It is well to remember that children are too young and usually too frightened to give an adequate and accurate history. The latter may often be said of the parents as well.

Treatment

The treatment of poisoning should be immediate. The child should be dispatched with the greatest expediency to a point, hospital or office, where treatment can be instituted immediately on arrival. It is of great value to have the parent bring the poison container. It may list the ingredients. Too frequently the anxious and overwrought parent forgets or destroys the container only to add additional time before definitive treatment may be started. The treatment may be divided into three stages: (1) removal of the poison; (2) administration of an antidote, and (3) supportive therapy.

Removal of Poison

The method of choice in ingested poisons is gastric lavage. In all cases or suspected cases of poisoning, lavage should be done immediately. There are certain exceptions to this rule. These exceptions are in patients who have consumed lye, ammonia, acids, strong alkali, and phenol. It is also to be avoided in the unconscious or convulsing patient. In the former group it is avoided because of the danger of perforation of the esophagus, and in the latter group because of the danger of aspiration during the procedure. It is the opinion of the author that lavage should be avoided in hydrocarbon (kerosene, fuel oil, and so forth) ingestion because of the increased incidence of bronchopneumonia following the procedure.

The technique of gastric lavage is a simple and well known one. With the child "mummied" or wrapped tightly in a sheet and placed on its side in Trendelenburg position, a mouth gag is introduced and the stomach tube passed. The larger the diameter of the tube that can be passed, the more easily and quickly can this procedure be carried out. Because of the possible and frequent occurrence of emesis during the procedure, suction should be available and used frequently to keep the nasopharynx free of mucus and vomitus. Using a small amount at a time, about 200 cc., the stomach is repeatedly lavaged with the solution of choice using a minimum volume of one liter. If the desired solution is not readily available, one should not delay the procedure. It is wiser to use water than to lose valuable time. Before the tube is withdrawn, the specific or universal antidote is placed in the stomach. The tube should be pinched before its withdrawal to prevent possible aspiration of material in the tube.

The use of emetics should be mentioned. In general, emetics should be avoided because they are usually inadequate to empty the stomach, and they tend to give one a false sense of security. They are usually difficult to get the child to take, and as a result much valuable time may be lost. If used, it should be only as a preliminary emergency measure and should be followed by lavage. Various emetics are available, but the one that is most readily available and effective is warm soapy water—dish water. Others that may be used are mustard water, milk, and egg white.

Antidotes

When one turns to one's therapeutic armamentarium in search for an antidote for a certain poison, one suddenly realizes that there is NOT an effective antidote for every known poison. As a matter of fact, specific antidotes are relatively few. Until the introduction of BAL (British anti-lewisite, 2, 3-dimercaptopropanol) there was no specific antidote for heavy metal poisoning. BAL is of value in heavy metal poisoning (mercury and arsenic) if administered early. It has no value if tissue damage has begun. BAL is dispensed as a 10% solution in peanut oil and is to be used for intramuscular injection only. The dosage is 2.5 mgm/Kg/ injection and given at four-hour intervals for twelve injections, then every six hours for four injections. BAL may produce toxic reactions such as lacrimation, salivation, nausea, vomiting, tachycardia, rise in blood pressure, and pain at the site of injection. The use of ephedrine sulfate in doses of 25 mgm. orally with each injection will help to offset some of these unpleasant side effects.

When no specific antidote is available, or when the nature of the poison is unknown, the use of the Universal Antidote is indicated. This should be placed in the stomach before the removal of the lavage tube. The constituents of this antidote are as follows:

Charcoal	2 parts
Magnesium oxide	1 part
Tannic acid	1 part

The charcoal will absorb the phenols and strychnine; magnesium oxide will neutralize the acids; the tannic acid will precipitate the alkaloids, glucosides, and some heavy metals. The function

TABLE I.

Poison	Common Source	Acute Symptoms	Treatment		
			Lavage	Antidote	Supportive
Strychnine	Cathartics, insecticides, rodenticides.	Central nervous system stimulant, hyperreflexia, opisthotonos, convulsions.	If no convulsion 1: 10,000 potassium permanganate.	No specific, use universal antidote.	Sedation, oxygen.
Arsenic	Insecticides, rodenticides, plant sprays, some paints.	Construction of throat, abdominal pain, projectile vomiting, diarrhea ➤, rice water stools ➤, bloody stools, convulsions and/or coma.	1% sodium thiosulfate.	BAL.	Morphine for pain, stimulants, treat for shock.
Lye	Draino pipe cleaner, toilet bowl cleaner.	Burning pain in mouth and stomach, mucous membranes soapy and ulcerated, bloody vomitus, collapse.	No and no emetic.	Dilute vinegar solution or lemon juice, olive oil for pain.	Fluids.
Sodium Hyposulfite	Sani-Flush, Clorox, bleaching agents, washing powders.	Vomiting, corrosive burns of lips, mouth, and tongue.	If no ulceration weak vinegar soln.	No specific.	Fluids.
Salicylates	Aspirin, oil of wintergreen.	Hyperpnea, listlessness, vomiting, dizziness, mental confusion, acidosis, hemorrhagic manifestations.	Milk or 1: 10,000 potassium permanganate.	No specific, instill saline cathartic in stomach after lavage.	Oral or parenteral fluids, watch electrolyte balance.
Barbiturate	Sedatives.	Somnolance ➤, stupor ➤, coma, respiratory and circulatory collapse.	If conscious.	Picrotoxin 1 to 10 mgm IV at 15 to 30 minute intervals. Instill saline cathartic in stomach after lavage.	Fluids, stimulants, oxygen.
Hydro Carbons	Kerosene, gasoline, naphtha, benzine, cleaning fluids. Fuel oil.	Burning pain in mouth, nausea and vomiting, drowsiness, confusion, fever. Aspiration is common ➤, bronchopneumonia.	No.	No specific.	Oxygen, fluids, antibiotics.
Fluoride	Insecticides.	Excess salivation, abdominal pain, hematemesis, shallow respirations—respiratory collapse.	Calcium chloride or milk.	No specific.	Fluids, calcium lactate IV.
Carbon Tetrachloride	Cleaning fluids, fire extinguishers, solvents for oils and fats.	Nausea and vomiting, headache, inebriation, convulsions. Late effect is liver necrosis.	1:10,000 potassium permanganate.	No specific.	Oxygen, fluids, high protein and carbohydrate diet.
Nicotine	Insecticides, plant sprays, tobacco, Black Flag.	Nausea and vomiting, mental confusion, salivation, abdominal cramps, convulsions, coma, respiratory failure.	Tannic acid or 1:10,000 potassium permanganate.	No specific.	Oxygen, stimulants.
Mercury	Antiseptics, fireworks, insect spray, also tablets.	Abdominal pain, vomiting, bloody diarrhea, circulatory collapse, kidney damage ➤, oligures ➤, anuria.	Copious amounts of milk.	BAL.	Fluids.
Analine Dye	Yellow and orange crayons, shoe polish	Apathy, dyspnea, cyanosis due to methemoglobinemia.	Water.	1% methylene blue IV (.2cc/Kg).	Oxygen, whole blood.
Ammonia	Household ammonia.	Burning in mouth and stomach, nausea and vomiting, abdominal pain, respiratory failure.	No.	Weak acids, olive oil.	Fluids.
Phosphorus	Rodenticides, roach poison, fireworks, imported matches*.	Nausea and vomiting, abdominal pain, diarrhea, shock, garlic odor to breath, liver and kidney damage.	2% copper sulfide then 1:10,000 potassium permanganate.	No specific.	Fluids.
Camphor	Camphorated oil, moth repellents.**	Odor to breath, headache, excitement ➤, delirium ➤, convulsions.	Water.	No specific.	Sedation, no opiates.
DDT	Insecticides.	Nausea and vomiting, weakness, vertigo, disorientation, coma.	Water.	No specific.	Fluids, sedation, Ca gluconat.
Cyanide	Rodenticides, metal polish, silver polish.	Odor of bitter almonds on breath, giddiness, headache, cyanosis, coma acts rapidly.	Early, 1: 10,000 potassium permanganate.	Immediate treatment may be of some value. Principle of treatment is to produce methemoglobin which in turn combines with the cyandie to form nontoxic cyanmethemoglobin. Inhale amyl nitrite pearl. Follow with 10cc of 10% Na nitrite IV.	

*Matches made in the U.S.A. are now made with insoluable phosphorus trisulfide and are relatively harmless.
**Many moth repellents now contain paradichlorobenzene which is relatively nontoxic.

of this antidote is to render the poison innocuous or unabsorbable. One cannot err in using this as an adjunct to all other therapy.

There are over 400 agents that have been known to cause acute poisonings. However, only a few of these are responsible for the majority of deaths. Although it is not the purpose of this paper to discuss the treatment of each poison, Table I outlines the most common poisons, their sources, symptoms and treatment.

Supportive Therapy

Close observation of all patients who have ingested poison is imperative regardless of the ini-

tial therapy. Hospitalization for at least a twenty-four-hour period is advisable, for delayed action such as shock, electrolyte imbalance, cyanosis, et cetera, may follow in spite of what may seem adequate therapy at the time. The judicious and propitious use of oxygen, blood, electrolytes, stimulants and analgesics may not only be lifesaving, but will also render the patient more comfortable.

Prevention

A discussion of poisoning in children is not complete without emphasis on prevention. It has been shown that accidents are now the greatest killer of our children. We have seen that deaths from accidental poisonings contribute significantly to this mortality. Of equal importance is the morbidity. A discussion of prevention of poisonings naturally overlaps into the field of all accident prevention in children. Significant advances in accident prevention have been made by such organizations as the National Safety Council, American Academy of Pediatrics, Children's Bureau, life insurance companies, and locally the Minnesota Department of Health. However, the campaign for the prevention of poisonings can be brought directly into the office of the individual physician. He can and should become the most ardent campaigner for this cause. He can awaken the parents to the possibility of poisonings in the home. He should lay the facts before the parent—the morbidity, the mortality, the serious sequelae. Emphasize the fact that 99 per cent of accidental poisonings are due to the neglect of the parents. A child cannot read the poison label. Specific instructions by the physician to the parents to carry out a household inspection on their arrival home will be rewarding. Have them open drawers, doors, cabinets in a search for "poison possibilities." The Minnesota Department of Health has available pamphlets and brochures for distribution to parents. This "gospel of prevention" should be given when the child begins to crawl, or preferably before. The physician may enhance the prevention program in another way. He can discourage the use of candy-like medications such as aspirin, and advise against use of over-the-counter and dubious home remedies.

The role of the physician in prevention is well summarized in the words of Dr. H. F. Dietrich: "If we don't accept this responsibility, remember that accidents may be the killers and parents may be the accomplices, but we the medical profession will be the accomplices before the fact."

A Poison Control Program

Although the primary responsibility of prevention and treatment of acute poisonings lies with the parents and the physician, the community can and should take an active part in such a program. A poisoning control program similar to that instituted by the University of Illinois for the city of Chicago could be constructed. The purpose of such program would be to furnish physicians and hospitals with current information on the diagnosis and treatment of poisonings. A current index could be compiled and maintained that would list the constituents of all proprietary drugs, patent medicines, home remedies, and household articles such as cleaning fluids, bleaches, insecticides, metal polishes, and so forth. This information would be available on the call of any physician or hospital. In addition to this, all cases of poisoning treated in the doctor's office, as an out patient in the emergency room, or in the hospital would be reported to this control center on a specially developed standard form. The compiling and analysis of this information would yield important data as to the type and frequency of poisoning. It would permit a better evaluation of treatment. It would allow when indicated, follow-up contacts to help correct those situations predisposing to the accident. It would expedite proper legislation to control the sale and distribution of poisonous materials. In addition to the foregoing, a poison control program would promulgate a campaign of prevention.

The nucleus of this program would be a committee composed of representatives—physicians when possible—from the city health department, state health department, department of pharmacology and toxicology of the University, department of pediatrics of the University, and of the department of internal medicine of the University. Until such time as a central treatment center for the community could be established, represented on this committee should be physicians delegated by staff of each hospital in the community. These physicians in turn would head the committee on poisonings in his respective hospital. In this manner all hospitals would be active participants in the control program. The above

(Continued on Page 897)

RAUWOLFIA

PAUL DE KRUIF
Holland, Michigan

IT WAS my first intention to make the title of this talk, "Rauwolfia—or how not to blow your top." But now it's just Rauwolfia. Your periodical, MINNESOTA MEDICINE, has asked me to submit the present talk for publication—and the vernacular in the first title would look bad in the table of contents, and might even scare the editors away from printing the story. For me there is a vanity that what I write for plain people might also be of some interest to the doctors. So, if this evening's entertainment turns out to be a bit stuffy, you now know why. Yet, it's going to be hard to be stuffy about this ancient and humble little shrub, *Rauwolfia serpentina.* The powder from its root is making miracles, even though they're slow and unspectacular. Rauwolfia is giving general practitioners a new, unexpected, unhoped-for chance to save life, to restore broken-down people to work, and, maybe, to prolong notably their span of productive vitality.

To this Rauwolfia hints a dividend. It gives family doctors a new weapon against anxiety. What all the psychoanalytic gobbledegook of neuropsychiatric specialists has had a hard time accomplishing, to the plain general practitioner Rauwolfia gives something powerful and tangible. Psychotherapy in pill form.

But forgive me, I'm getting ahead of my story. What originally excited me about Rauwolfia was an article by Ford and Moyer, of Houston, Texas, in GP published in November, 1953. It told how Rauwolfia has at last put the treatment of high blood pressure—including severe forms of it—into the hands of general practitioners. Four years ago this was beyond the power of the smartest internists; and the stakes the family doctors are going to play for in this particular grim game of life-or-death, are high—a fifth of all deaths in our country are due, directly or indirectly, to the consequences of hypertension.

And now Rauwolfia is going to put plain GP's into the front line in such a fight? With real power? What a story.

Yes, and what a tough story to write. When you set out to peddle hope among 17,500,000 people buying the Reader's Digest, you'd better be sure. And here's the trouble. Hypertension, of course, *can* be harmless; many people live out a normal life span unaware they have it. And there are big-shot internists, notably in New York, who'll tell you of old ladies—pressure 210/120 for years—living hale and hearty till eighty-five. Then dying—of cancer, not hypertension.

Certain of these big clinicians believe it best hypertension be kept a secret from their patients. Out of one side of their mouths these internists imply high blood pressure isn't going to kill you. Out of the other they say knowing you've got it will scare your pressure higher, and that might be dangerous. This confused me.

Then too, what about this new-fangled, old drug, Rauwolfia, and the new treatment it makes possible? In 1953 the eminent *Lancet,* of Great Britain, editorialized that chemical treatment of hypertension was so difficult that clinicians could still do no more than keep their patients' pressure a secret.

On the other hand there was information—grim and reliable—that people with essential hypertension with onset at age thirty-two, on the average only lived till fifty-one. Then dying—of the consequences of high blood pressure. Now at Reader's Digest, if you want to get a story into it, there's one thing you'd better not be, and

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Presented at the annual meeting of the Northern Minnesota Medical Association, Willmar, Minnesota, September 10 and 11, 1954.

EDITOR'S NOTE: The editor considers it a great privilege to publish an article written by Paul de Kruif, dean of medical science writers. In spite of the widely-experienced irritations due to inquiring patients stimulated by his many contributions in the lay press, physicians have learned and gained much from his writings. There is probably no single person in our time who has presented the benefits of good medical care to the public more ably than Paul de Kruif.

Paul de Kruif is roving editor for *Reader's Digest.*

that's confusing. The project seemed stymied before it got started. What to do?

It was my good luck to find a couple of clinical investigators in the hypertension field who talked not out of both sides but straight out of the middle of their mouths. These two fellows—whose discoveries will make the burden of the story—became my briefing-officers. And now, physicians that you are, you must bear with me if I talk very simply.

Forgive me, too, if I digress for a moment to explain why I'm going to stick to A B C's. At a welcome Travelers broadcast, not long ago, the M.C., Tommy Bartlett, asked me: "How come you can write complicated medical science to millions of people?"

"Only because I'm so dumb," I answered.

"What do you mean dumb?" asked Tommy.

"To get to understand that highbrow stuff," I said, "being really dumb, I've got to boil it down to the littlest words. When it's clear to me, dumber than dumb, it'll be clear to *anybody*, no matter how low their I. Q."

So, in what follows, forgive me if I talk up to you instead of down to you. Getting going on the investigation for the story, I boiled down heavy scientific publications to the simplest questions, asked of my briefing-officers.

"If so many can live years, not knowing they're hypertensive, how can you fellows be sure a given case is dangerous?" I asked them. They both agreed they could not be, but there were clues. It's more likely to be serious when it runs in families or starts early in grownup life.

"How do you diagnose serious hypertension?" I asked them. That was tough, they agreed. It starts insidiously. Only a minority have early symptoms (notably fatigue) sending them to a doctor. Most hypertension is stumbled on at insurance, and other routine examinations.

But what makes high blood pressure dangerous? My briefing-officers explained the heart has to pump harder against a chronic, rising pressure. Till at last its muscle may fail. And when the pressure stays high, for years, it *aggravates* thickening and deterioration of arteries. If the heart's arteries plug up, there is a coronary accident; if the brain arteries plug or burst, there's a stroke; and damage to kidney arteries may result in fatal uremia.

This sinister trinity, my teachers said, is the king-killer, responsible for the death of 320,000 Americans yearly.

Then, in my simplicity, I popped my $64 question—

"What *is* it that makes blood pressure go up so dangerously?"

One of my briefing-officers, Dr. Robert W. Wilkins, of Boston, tried to explain it. "It's a complicated mechanism," said Bob, "involving the sympathetic nervous system, endocrines, muscle tension, a possible toxin from the kidneys." Bob's words were pretty big. "Give me a handle to hold on to," I asked.

Well, one factor was clear, explained Bob. A great majority of cases start from deranged nerve cell activity causing a chronic spasm of the blood vessels. Overactive impulses go from deep in the brain out over the sympathetic nervous system, which, independently of our will, controls organs, glands, blood vessels. These super-active nerve impulses narrow down the arterioles, the little valves at the ends of the arteries; so the heart's got to pump harder to circulate the blood, and so shoots up the pressure.

My other briefing-officer, Dr. John Moyer, of Houston, Texas, backed up Bob Wilkins. "We don't know the underlying cause of hypertension," explained John. "But a common denominator is over-activity of the sympathetic nervous system."

"How do you know?" I asked. "Because when you interrupt the outflow of those nervous impulses, down goes the pressure," said John.

"Therapeutic nihilists may say high blood pressure in itself doesn't kill anybody," said Bob Wilkins. "But that's a quibble, like saying, does a man die of too much acid in his stomach when he has an ulcer, or does he die because acid causes the ulcer and makes it bleed and he dies of bleeding to death."

"I don't get the analogy," I said.

"Well," said Bob, exasperated. "High blood pressure doesn't kill you, but when you have it year after year, it *does something* to you. Your blood vessels get so shot, they can't stand the pressure, in the heart or brains or kidneys, so you say 'I'm not dying *directly* of high blood pressure, I'm only dying of the *effects* of high blood pressure.'

"It's a damned quibble," said Bob.

If John Moyer and Bob Wilkins are right, if the sympathetic nervous system was in part to

blame for high blood pressure, why not cut its nerves, blocking overactive impulses?

That question flashed my memory back to what was, for me, a momentous day in 1933. That day the late great Dr. Max Minor Peet, neuro-surgeon at the University of Michigan, excited like a boy as he always was about his work, told me how he'd just asked that question, and then acted on it. He risked a new operation, cutting out the thoracic ganglia of the sympathetic nervous system of a man terribly hypertensive and about to die. The patient's pressure had dropped to normal, his vision returned, he was back to work, as good as dragged from the grave.

That patient worked, in good health, for many years, outliving Max who had saved his life. Max Peet had proved the sympathetic nervous system was not necessary to life—and that was a break for doomed hypertensives. He'd proved the basic fact that there's a neurogenic factor in hypertension. His now famous sympathectomy operation—extended and made more drastic by Dr. Reginald Smithwick of Boston and Dr. Keith Grimson of Durham, North Carolina—has saved thousands of hypertensives threatened with early death.

Yet for millions of *not yet desperately ill* hypertensives, sympathectomy was not the answer. Many wouldn't face it. Too risky. They preferred to take their chances. Many kept dying.

Then came a synthetic drug from England by way of New Zealand; hexamethonium, blocking the sympathetics, chemically. It knocked all high blood pressures, no matter how terrific, down to normal. From Switzerland from Ciba Laboratories came hydralazine. Both chemicals were sensational . . . too sensational.

In 1950 Dr. Edward D. Freis, in Washington, D. C., and Dr. Henry A. Schroeder, in St. Louis, combined hexamethonium and hydralazine into a double-barrelled cannon. To shoot at the sympathetic ganglia of malignant hypertensives, who were going to be dead in a year, or not much more. Why did Freis and Schroeder give the new treatment such a terrible test?

Because the new treatment, too, was superdrastic. Hexamethonium—"dangerous hex" it came to be called—drove the highest blood pressures down so low so fast that patients often fell on their faces, flopped on the floor and passed out cold. Hydralazine caused severe headaches, nausea and fast pounding of hearts already likely to be damaged.

But Freis and Schroeder, like doctors everywhere, helpless, had watched malignant hypertensives die. So they risked it. Out of 100 of Schroeder's malignant hypertensives, 63 have survived from one to two years. Pressures? Back, practically, to normal. No further damage to hearts, brains or kidneys; 57 back working. Freis's results were similar.

But these resurrected ones lived under a sword; 11 stopped treatment; within a few weeks all were dead. If only they'd stuck with it? But sometimes it was too tough to take.

Hexamethonium-hydralazine was only for patients in hospitals and under close supervision of experts. For many it was too high a price to pay for living; it was definitely no medicine for busy practitioners to use on hypertensives, generally.

Then out of the mists of Indian oblivion arose Rauwolfia. For hundreds of years the powdered root of this lovely shrub with its bright green leaves and pinkish-white flowers had been hawked in the bazaars as a cure for lunacy, diarrhea and snake bite. It is reliably reported that Mahatma Ghandi, by chewing on Rauwolfia root, gained the calm that drove the British so wild that they went away, without a struggle, giving India back to the Indians.

In 1950, Dr. Robert W. Wilkins, of Boston, read an article by Parsee Dr. Rustom Jal Vakil who said that Rauwolfia was good for hypertension, too. Wilkins was not so God-Almighty that he couldn't take a tip from beknighted Asians. Wilkins ignored the crazy claims; and tried Rauwolfia—on hypertensives.

Just as all previous hypertensive drugs had been tested, he gave it in single, trial doses, bigger and bigger. Nothing happened. Now all really powerful blood pressure-lowering chemicals act quickly, if at all. Wilkins nearly threw Rauwolfia out the window as just another one of those things.

But one of his patients, a Negro lady, Carrie Brooks, she should be historic, ***happened,*** repeat ***happened,*** without design, to be given Rauwolfia pills not once, but five days in succession. Did a resident misread directions on the chart? We don't know. But on the sixth day her extremely high, fixed blood pressure was down to normal. That accident started Rauwolfia on its American adventure.

Wilkins was astounded by Rauwolfia. It took three to six days of q.i.d. doses to show any effect at all. It took from three to six weeks to reach peak activity. After it was stopped, it was one to three weeks before its effects wore off. In all pharmacology, this was unprecedented. In contrast to other chemicals that knocked blood pressure down violently, Rauwolfia was gentle. It lowered pressures, modestly. It brought only thirteen of Wilkin's first thirty-nine cases down to normal.

Rauwolfia was really uncanny. A physician brought one of his patients to Wilkins. She was severely hypertensive—pressure 250/150—banging occipital headaches—ready to blow her top—E.K.G. signs of heart strain—veratrum lowered her pressure a little, but at the expense of severe vomiting. At Wilkin's suggestion this woman's doctor added Rauwolfia to the nauseating veratrum.

Over months, her pressure started slowly down. Soon after that, veratrum could be cut to doses that were not nauseating. That was in 1950. Today this businesswoman is at full activity, blood pressure normal, on small daily doses of veratrum and Rauwolfia.

There was a weird side-effect of Rauwolfia on this lady—it changed her personality. She reported: "I just don't seem to have to do the things I used to. Before Rauwolfia, I *had* to do things. Now I don't care. Dust doesn't annoy me anymore. Before, I knew it wasn't important, but even though tired, I had to clean it up the moment I got home from work. Now I can let it go till next day."

It happened that this lady detested her husband so that her own doctor refrained from mentioning his name because of its immediate effect on her dangerous blood pressure. Wilkins made an experiment. He got her going on her husband's sorry characteristics. Under Rauwolfia's gentle sway, she remained calm as a May morning; her pressure stayed normal.

Among hypotensive drugs, Rauwolfia is exceptional. It is no blocker of the sympathetic nervous system. It acts on emotions, in their power-house, the hypothalamus, in the bottom of the brain. It's more than a mere sedative. In already heavily sedated, even anesthetized dogs, it lowers their blood pressure and slows their hearts.

On hundreds of patients, Wilkins has watched the Rauwolfia effect; and it's no doper-upper, like phenobarbital. It's unique in medicine—a tranquilizer. And it makes up for its modest lowering of blood pressure by an amazing effect on hypertensive symptoms. Rauwolfia soothes the headaches, dizziness and wooziness, pulls butterflies out of the stomachs of anxious neurotics. It's no soporific. Patients tell Wilkins: "It doesn't put me to sleep but it lets me go to sleep, if I'm sleepy."

Wilkins observed that, though it calms people, it doesn't turn them into zombies. Patients under Rauwolfia reported they might go to sleep in church if a sermon was dull, but were right in there, watching a prize-fight on TV.

Bob Wilkins discovered another trait of this strange Rauwolfia. It had a way of taming the bad effects of the more powerful hypotensive drugs that still had to be used against severe hypertension. It slowed down hearts pounding hard and fast from hydralazine (Apresoline). In other patients besides that businesswoman, it cut down veratrum to non-nauseating doses still effective against high pressure.

Wilkins rejoiced to find that Rauwolfia's own side-effects were trivial. Some stuffy nose, dreams more vivid than usual, overactive intestinal elimination, some depression when doses were too high; all reversible by withholding the drug for a while or cutting the amount of it down.

Bob Wilkins is one of those clinical investigators who does not live in an academic world of his own; he is always looking for treatment usable by general practitioners. "Don't expect dramatic results from Rauwolfia," he kept telling them. "Wait for several weeks or months to judge its effectiveness."

Here was another merit of Rauwolfia, important to the family doctor. Often less and less of the more powerful, dangerous hypotensive drugs could be given, when they were bolstered by gentle Rauwolfia. Till at last, this safe Rauwolfia, all alone, in certain cases though not in all, held pressures down to normal.

Aside from these qualities, another action of this mystical Indian powder haunted Bob Wilkins. For example an executive, his formerly very high pressure now normal, on Rauwolfia alone, reported—"There are things that in the old days, bothered me. Now I refuse to let them disturb me. Last year a man would make a mistake at the factory and I'd blow my stack. Now I just show

him the right way to do it. This stuff is fabulous!"

This executive's wife was amazed at the change in her man. "He used to get furious, cursing out bum drivers on the road. Not any more. What's *happened* to him?" she asked.

How far did the power of Rauwolfia extend? Surely this Indian medicine, so gentle in its action, wouldn't be able to tame the most powerful of all hypotensive drugs—hexamethonium—dangerous hex—that, alone, could bring super-high pressures down, when all else failed? But dangerously, too dangerously.

Dr. John H. Moyer of Houston, Texas, put Rauwolfia to this sternest of all tests, on a forty-seven-year-old woman who was having a rugged time, trying to stay alive, on hexamethonium alone. One year before, dangerous hex had rescued her, in coma, near blind, in hypertensive crisis and about to die, blood pressure 300/200.

Hexamethonium shot her deadly pressure down to normal and way down below normal till almost no pressure at all. "Get your wife away from those Baylor University docs, they're goin' to kill her," their family physician told the lady's husband. But her husband overruled the doctor. He knew her banging headaches were gone; she was home; she could see again; at least she was alive.

But now John Moyer added Rauwolfia to the dangerous hex. The wild up-and-down swings of her pressure steadied. In a few weeks she needed only half of hexamethonium's former dose to keep her blood pressure close to normal. In a few months she no longer needed dangerous hex at all. Just Rauwolfia, a few safe tablets daily.

It was my honor to meet this patient. I have never seen a lady more serene, relaxed, more obviously healthy and lovely—three years after she should have been dead.

Among twenty-seven similar surely doomed people, brought out of hypertensive crises by combined Rauwolfia-hexamethonium, laboratory tests have given the Moyer group evidence of arrest, and actual reversal of deterioration of the arteries of the heart, the eyes and brain.

At the Jefferson Davis Hypertension Clinic, Moyer and his co-workers have tested hundreds of hypertensives—most of them far more severe than the average found in private practice—in parallel treatment series, using Rauwolfia alone or in various combinations with hydralazine, veratrum or hexamethonium. They report blood pressure significantly lowered in nine out of ten; brought down to normal in nearly half; and seven out of ten, incapacitated, are back at work.

What about the very early, mild hypertensives, who are predominantly seen by general practitioners? Moyer reports 63 per cent of these respond, when kept on Rauwolfia alone, for more than a year, now. Even in early but moderately severe hypertensives who respond initially, Rauwolfia for a year has kept their blood pressure normal in forty out of fifty. In the remaining ten whose pressures broke over a bit, they were quickly and easily brought back within bounds by safe, moderate doses of the more powerful hypotensive drugs, added to Rauwolfia.

Bob Wilkins points out that these results open a whole new vista to general practitioners, who alone see this twenty-year disease at its start when it's symptomless or mild. Now the family doctor has a golden chance to prevent essential hypertension from becoming serious, and terminating fatally, as has heretofore been inevitable.

Now that they can safely treat early high blood pressure in their offices, general practitioners can start their own preventive war against this king killer.

In a recent issue of the *Journal of the American Medical Association*, John Moyer sums up the promise of the Rauwolfia revolution, as follows: "One has every reason to believe the disease may now be controlled in its incipiency; that serious complications may consequently be prevented; that combined drug approach to management of severe cases may prevent their progression; and that it may reverse previously inevitable advance of co-existent vascular disease."

Robert Wilkins points up Rauwolfia's sudden upset of the sad hypertension situation: "Less than five years ago when we were asked to lower serious hypertension with drugs, we had nothing to offer," he writes. "Now no case can be called impossible to treat until so proved."

Of the millions of Americans for whom these words may mean a new lease on life, all—except a possible ten per cent of malignant hypertensives who must be hospitalized—can be treated by general practitioners in their offices. If—and I'm speaking to you doctors now—this can be done, it can be done only if you will take the time to learn this not too difficult science. And you alone,

as personal physicians to your patients can follow the results.

And this brings us to Rauwolfia's dividend, its curious effect on anxiety. Again and again during the past five years Bob Wilkins has been struck by this eerie medicine's transformation of beat-up hypertensives. Many told Wilkins—

"I've *never* felt so well," or "This is how I *dreamed* of feeling," or "*Nothing* bothers me now" or "I just don't *give* a damn."

What was the common denominator of these testimonials to Rauwolfia? It wasn't that the patients were no longer hypertensive. It was that they were no longer *anxious*. And if anxiety is a seven-letter-word for the four-letter-word, fear; and if fear is an emotion; and if emotions arise in that little nest of nerve cells in the hypothalamus at the bottom of the brain; and if—as it seems from experimental pharmacologic evidence —Rauwolfia is a hypothalamic regulator—then why not try Rauwolfia on anxiety? Pure cases of anxiety, devoid of high blood pressure?

Again, the benighted Hindus had scooped Bob Wilkins on his intuition. And not Hindu doctors either, but anonymous low-down laymen had antedated him. For hundreds of years, Rauwolfia, under the people's name for it, "Pagal-Ka-Dawa" had been sold in bazaars—across-the-counter—for insanity. Utterly unscientific. Pure folk medicine. Then the Hindu Dr. Hakim, a few years ago, proved his lay brothers had something.

Then Rauwolfia went highbrow on its anonymous discoverers, went scientific, became medically respectable. In Basle, Switzerland, Ciba chemists crystallized a pure alkaloid, reserpine, out of the yellow powder of the crude root of this Pagal-Ka-Dawa which means "insanity herb" to Hindus.

And these reserpine crystals can be injected by vein or into muscles of human beings, safely, in doses huge compared to those used in hypertension. And at Modesto State Hospital in California, this was done, last year, by Drs. Robert H. Noce, David B. Williams and Walter Rapaport.

Their first human experimental animals were hopelessly psychotic, picked from what are called "maximum security wards," sinister halls in continuous turmoil due to raucous, hyperactive, combative, sarcastic, resistive, uncooperative patients.

Then by injection and by mouth, into this cohort of the stark, staring, raving insane, went reserpine, trade name, "Serpasil," pure alkaloid of Rauwolfia.

Over the bedlam of the maximum security ward came a change. In this "back ward" of the madhouse there began to reign, tranquillity. Noisy patients became quiet and calm. Depressed patients became alert and cheerful. On the Serpasil wards, restraint, sedation and electroshock have been almost eliminated. In 80 per cent of patients the improvement was real, even striking. Already, 20 per cent of those hopeless psychotics are in full remission and eight have been discharged; and the longer the treatment is maintained, the greater the number of remissions.

A report on this wôrk has been accepted for publication by the *Journal of the American Medical Association*. The effect of Rauwolfia on the mentally disturbed is under study, nationwide, by more than 100 investigators.

Along with the remarkable new synthetic chemical, chlorpromazine (that's another and maybe a similarly hopeful story) reserpine means hope for many of the insane.

But psychotics weren't Bob Wilkins' worry. His concern is for the simply anxious. Not the anxiety that can be treated because it's due to malnutrition, Brucellosis or this or that vitamin or hormonal deficiency. Wilkins was thinking of the army of neurotics with worries that psychotherapy may suppress but can't cure; neurotics who are still anxious, after it's been explained to them, as they lie on a couch in a darkened room, once a week at so much an hour for two or three years, that their anxiety is due to having been in love, as infants, with their papas or their mamas.

Bob Wilkins' interest is with pure, seemingly causeless anxiety, with those neurotics who explain to their doctor they're worried because they haven't a thing to worry about, saying "What I'm worried about is really nothing. So there must be something wrong with my mind."

Would Rauwolfia or its pure alkaloid, reserpine, put a chemical block into this vicious circle? On a growing number of these anxious neurotics Wilkins and many other physicians are testing it; successfully, in many. They're finding that Rauwolfia is a drug against fear.

This is a new challenge to general practitioners who are fighting, not too successfully, with

(Continued on Page 903)

THE DESMOID TUMOR

Two Case Reports with Methods of Repair

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DESMOID TUMORS are benign fibrous tissue neoplasms which arise from the musculoaponeurotic structures throughout the body, and have the significant feature of locally invading adjacent muscle tissue and engulfing such tissues with actual replacement of the striped muscle fibers. The first description of this condition was in 1832 by MacFarlane[6] and in 1848 Mueller[7] coined the term "desmoid" from the Greek words "Desmos" meaning a band or tendon and "Eidos" meaning appearance. Thus the actual derivation of the word comes from tendon-like.

Location

As stated in the definition, these tumors can be found almost anywhere that striated muscle is found or in its aponeuroses. The most frequently reported locations are, however, in the abdominal wall musculature and the tumors are most commonly seen in relation to the rectus muscle below the level of the umbilicus. It has been stated by Stone[10] that the right lower part of the abdominal wall is involved more frequently than any other location.

Incidence

Desmoid tumors are somewhat of a rarity. This can be shown by the fact that Musgrove and MacDonald[8] found only 120 cases in the files of the Mayo Clinic from 1906 through 1945 inclusive. Of these, eighty-five were abdominal desmoids and forty-five were located elsewhere. Labbe and Remy[5] stated that Bilroth saw only sixteen abdominal desmoids in sixteen years. Stone[10] reported only five desmoids in 21,000 surgical patients at Johns Hopkins Hospital. Pack and Ehrlick[6] encountered only seventeen desmoid tumors of the abdominal wall from 1917 to 1943 in 50,346 patients with neoplasms. This was an over-all incidence in their series of 0.03 per cent.

This tumor is most commonly seen in women during the child-bearing years from twenty to forty, whereas in males such growths, though very uncommon, usually come on later in life. These tumors have been very rarely seen in childhood and one congenital case was reported by Brockman.[1]

Etiology

Muscle trauma is considered to be the chief factor in the development of the desmoid tumor. Such injuries as muscle tears and stretching, which occur during pregnancy and labor, offer an explanation for the occurrence of these tumors during or shortly after pregnancy. The tumors also occur in old surgical scars, also showing trauma as being basic to their etiology. A patient was described by Green[4] in which a desmoid tumor developed in the inguinal canal of a patient, who had worn a truss for a period of ten years.

It has been suggested in the literature that there is a relationship between sex hormones and desmoid tumors. Geshickter and Lewis[3] have described a similiarity between desmoid tumors and fibromyomas of the uterus as well as fibroadenomas of the breast. They found that all three of these tumors have a special ability to concentrate pituitary gonodotropin substance and estrogenic substance, and mentioned that a biologic assay of one desmoid tumor yielded 13,000 rat units of gonadotropin per kilogram.

Pathology

Clinically, desmoid tumors are somewhat similar to fibroid tumors in other locations, except that there is no definite capsule. These tumors are generally described as being densely hard, rubbery in consistency and cut with a grating sound. Small to medium size tumors usually are of whitish-pink in color and appear somewhat glistening on cut surface. The larger tumors may show areas of degeneration due to lack of central blood supply and may be somewhat differ-

ent in color. There are numerous interlacing bands of fibrous tissue throughout the tumor and the periphery of the cut surface of the tumor shows inclusion of bundles of muscle fibers.

The microscopic appearance is that of a cellular fibroma occurring in striated muscle. According to Ewing,[2] the tumor structure may resemble a neurofibroma and may vary from a hard somewhat cellular fibroma to a rather cellular fibrosarcoma. However, Musgrove and MacDonald[8] do not agree with this view, and feel that these tumors are all benign and recur only locally due to inadequate excision. Thus, in their careful study of a group of desmoids and a group of fibrosarcomas, they felt that the two are completely separate entities. They do admit, however, that the border line cases are very difficult to distinguish. Histologically the picture is that of a cellular fibroma with interlacing whorls and bundles of fibrocytes. Fibroblasts in the periphery of the tumor appear to be more active and seem to be invading the surrounding striated muscle, and some of the surrounding muscle fibres frequently undergo degeneration. Occasionally giant cells may be seen.

The tumor does not produce distant metastases but is locally invasive and recurs frequently unless adequately excised. Pack and Ehrlick[9] reported one tumor as having recurred six times after surgical removal. On occasion the tumor may invade bone and peritoneum, but skin is involved only by ulceration due to pressure necrosis. The largest desmoid tumor ever recorded weighing 17 kilograms was reported in 1850 by Rokitanski.

Symptoms and Signs

Due to the usual location of these tumors they do not produce much in the way of symptoms. The patient frequently notices a small hard nontender mass in the lower abdomen and may come to the doctor because of it. Some of the larger tumors may cause mild sensation but in general these tumors do not cause pain. On palpation the tumor is felt to be smooth in contour but somewhat hard in consistency. Generally the skin can be moved freely over it. In order to differentiate between an intra-abdominal tumor and one within the abdominal wall one must use a special maneuver which calls for examining the abdominal muscles first when they are tightly contracted and then when they are relaxed. The patient is asked to sit up from a lying down position and this causes the abdominal muscles to contract which in turn causes a mass within the abdomen to become indistinct whereas a mass within the abdominal wall becomes more prominent. When the abdominal muscles are relaxed the tumors are more freely movable particularly in the plane perpendicular to the course of the muscle fibres to which they are attached. The main thing in the diagnosis of this quite rare condition is to remember that such a condition exists.

Differential Diagnosis

When one finds a tumor mass which conceivably could fit the above description, one must think of several different types of conditions to make the diagnosis. There are intra-abdominal tumors such as tumors of the caecum, uterine fibromyomas, and tumors of the ovary. If any of these become attached to the anterior abdominal wall they may simulate desmoid tumors. Any tumor arising from bone which might invade the abdominal wall could give the appearance of a desmoid tumor. Sometimes hernias may simulate desmoid tumors. Occasionally, tumors such as neurofibromas and lipomas may simulate desmoid tumors. Generally, one comes back to the old adage that "the only good place for a tumor is in a specimen bottle."

Treatment

Complete surgical removal with a wide margin of normal tissue on all sides of the growth is vital to the treatment of this condition. Therefore, it is essential to think of the condition and recognize it at surgery so that such an excision can be made. Although it is possible to enucleate the tumor in some cases, one is not actually removing the whole tumor, but is leaving portions of it, since this is not an encapsulated growth. Frequently these tumors require a wide excision of tissue leaving a rather extensive defect. Sometimes these defects may be closed without undue tension but it frequently is necessary to use some form of re-enforcement tissue such as facial transplants, etc.

There has been some evidence to show that x-ray and radium may have a role in the treatment of cases considered inoperable due to extensive growth or poor risk patients. However, this is true only in females since desmoid tumors in males are definitely radio-resistant. This latter

fact makes one assume that castration or radiation therapy of the ovaries might have some significance in the treatment of desmoids in women. Pack[9] was able to cause a desmoid tumor of the shoulder to regress by irradiation castration of the ovaries as the only treatment. Apparently there has been no literature describing the use of sex hormones in the treatment of this condition.

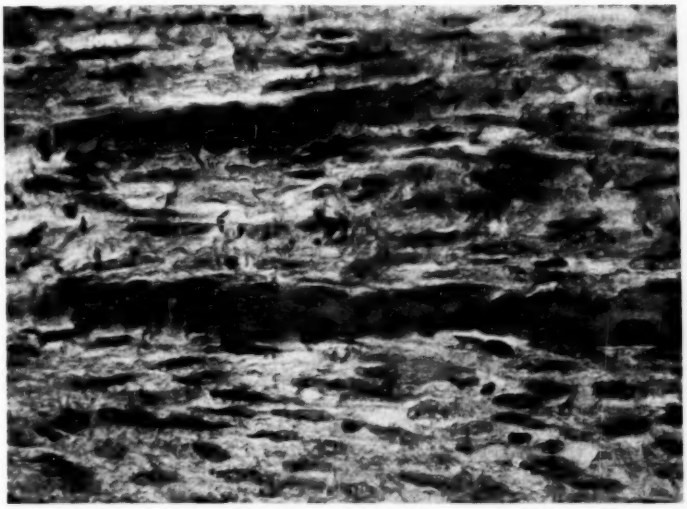

Fig. 1. High power photomicrograph showing the moderately cellular fibrocytic neoplasm invading striated muscle.

In reviewing the existing records at St. Mary's and St. Luke's Hospitals at Duluth, Minnesota, only two cases of desmoid tumors were found.

Case Report

Case 1.—A twenty-seven-year-old white woman, first seen on March 27, 1950, came in because she had noted a lump in her right lower abdomen in the region of the upper portion of an old appendectomy scar. The appendectomy had been performed in 1946. Although she had noted some slight discomfort in her right lower abdomen off and on, for several months, she had not noticed the lump until a few days before she came in. She stated that the discomfort was more noticeable a few days before each period and that this had been noticed during the past three to four months prior to her visit to my office. She stated that the discomfort consisted of a slight burning sensation and was aggravated when she wore a girdle.

The patient's past history was negative for any significant illnesses or injuries and she had never been pregnant. The general physical examination was negative except for the mass which was located in the lower portion of an old right rectus scar. The skin was movtive except for some tenderness deep in the cul-de-sac. Routine laboratory examinations were essentially normal including an x-ray study of the colon.

In the differential diagnosis of this case, the tumor was considered to be arising from: (1) The abdominal wall musculature, (2) The caecum, (3) Ovarian tumor, (4) Endometriosis inplanted in the abdominal wound following an abdominal operation.

On April 17, 1950, operation was performed under sodium pentothal anesthesia and a right rectus type incision was made directly over the lesion. On opening the fascia over the right rectus muscle, it was found that the mass was adherent to the anterior rectus sheath as well as originating in the muscle below. Resection of the tumor with an area of what was thought to be normal tissue and fascia around it was carried out and the defect was closed in routine fashion. The report by Dr. Arthur Wells, pathologist, revealed that the specimen consisted of firm tumor nodule measuring 4 cm. by 3.5 cm., which on cut section had firm consistency and a somewhat grayish refractile cut surface. Microscopic study revealed trabeculae of fibrocytes of moderate cellularity which invaded the attached muscle causing atrophy and regenerative changes in muscle cells (Fig. 1). He stated that the invasion extended to the line of excision and recommended a wider excision of tissue. Accordingly, on April 20, 1950, the patient was brought back to the operating room and a resection of most of the right rectus muscle and its

fascia from the umbilicus to the pubis was performed leaving the peritoneum intact since it was not involved. A piece of tantalum mesh gauze about two and one-half inches wide by five inches long was placed in this defect and imbricated up under the remaining lateral and medial borders of the anterior rectus sheath with interrupted sutures, and the abdomen was closed in routine fashion. The pathologist reported that the excision included all the tumor tissue, and the patient has had no recurrence of the tumor to date. As evidence of the strength of her abdominal wall following this procedure, she successfully went through a pregnancy and delivered a normal infant approximately two years after her original operation with no evidence of further weakening of the abdominal wall.

Case 2.—A seventy-year-old woman is included in our review through the courtesy of Dr. Robert LaBree of the Duluth Clinic. The patient had a cholecystectomy approximately three years previously. She noted a small bulging in the lower portion of the incision shortly thereafter and this subsequently continued to enlarge. About a year prior to her hospitalization for the present condition she had noted a firm mass in the upper portion of the incision which had gradually enlarged. She noted discomfort in this region which at times had actually been painful. Examination of the abdomen showed a fixed mass 4 cm. in diameter at the upper end of a right upper quadrant scar. A hernia was present in the lower portion of the same scar. There were no other palpable masses or tenderness. There is no mention in the history as to whether the patient ever had children.

On January 9, 1951, the patient had a repair of ventral hernia with excision of the mass in the upper end of the hernia. In order to excise the mass so much of the fascia was removed that the surgeon decided to re-enforce the repair with a cutis split skin graft. She had an uneventful post-operative recovery. The pathologist reported that the tumor mass measured 8 by 6 by 5 cm. and on cut section was of diffuse fibrous consistency throughout and somewhat gray in color with no evidence of hemorrhage or caseation. The microscopic study showed that the tumor mass was moderately cellular and was made up of fibrocytes arranged in the regular interweaving trabeculae. There were very rare mytotic figures. The cells appeared mature. The multiple sections of the outer margin revealed no invasion beyond the compressed adjacent tissues. Diagnosis: "Fibroma of the abdominal wall consistent with desmoid tumor."

The pathologist added this note: "The histology of the lesion is perfectly satisfactory for a diagnosis of either a fibroma or desmoid. The fact that there is no infiltration of the adjacent connective tissues or muscle favors the benign lesion. However, the patient should be given the benefit of the doubt and any suspicion of re-occurrence should be immediately investigated and if proved to be a re-occurrence, a much wider excision into normal tissue should be carried out." In checking with the surgeon, the patient has had no recurrence up to the present time.

Summary

The literature on desmoid tumor has been briefly reviewed. A fairly typical case has been presented of a desmoid tumor arising in an appendectomy scar in a young woman several years after the surgery had been performed. A second case of desmoid tumor arising in an older woman has been reviewed.

The use of tantalum mesh gauze for re-enforcing the abdominal wall defects following the excision of large portions of the abdominal wall for removal of desmoid tumors has been presented.

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SUBCUTANEOUS EMPHYSEMA FOLLOWING TONSILLECTOMY AND ADENOIDECTOMY

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WE WISH to report an unusual case of subcutaneous emphysema of the face, neck, and thorax following tonsillectomy and adenoidectomy. Although the patient recovered, the sudden appearance of widespread subcutaneous emphysema caused undue alarm among the physicians attending the patient, and we wish to bring the condition to the attention of other physicians who may encounter this unusual, but not rare, complication.

Case History

A six-year-old boy was admitted to St. Luke's Hospital on June 10, 1954, for tonsillectomy and adenoidectomy. Past history was negligible except for numerous sore throats and bouts of acute tonsillitis. The physical examination revealed enlarged tonsils. Laboratory studies of the blood and urine were normal. Preoperative medication consisted of codeine sulfate gr. ½ and atropine sulfate gr. 1/400 given subcutaneously forty-five minutes before the operation.

Induction of anesthesia was begun with open drop vinethene and was maintained with diethyl ether. An endotracheal tube (soft-rubber King No. 4) was inserted into the trachea without difficulty and the endotracheal tube was connected to a Heidbrink anesthesia machine which contained a functioning pop-off (expiration) valve. Nitrous oxide, 2 liters per minute, and oxygen, 2 liters per minute, were administered with ether during the course of the anesthesia. The pop-off valve functioned throughout the course of anesthesia. Tonsillectomy was performed using the Beck snare technique. During the adenoidectomy the bleeding was greater than usual and an adenoid pack was inserted. At the completion of the operation the patient was turned on his abdomen with the operating table in a 10 degree Trendelenburg position, and was allowed to emerge from the anesthesia. When he began to gag on the endotracheal tube, the tube was removed. It was noticed that the patient was still bleeding from the nose and therefore two cigarette packs were inserted into the nostrils, packing off the nasal cavity tightly. The patient was then taken to the Recovery Room where he vomited and coughed more violently than is usual. After about fifteen minutes the nurse noticed a swelling of his left eye. The left eyelids were swollen completely shut and the left cheek was markedly enlarged. Crepitation was felt in the swollen areas as well as on the forehead, neck, supra-clavicular area, and in the skin over the anterior chest wall (Fig. 1). The patient breathed normally and denied having pain from the swelling. X-ray examinations in the posteroanterior and lateral projections showed subcutaneous enphysema involving both sides of the neck with anterior mediastinal extension of the process. Following consultation with a chest surgeon, it was decided that since there was no respiratory embarrassment, observation of the patient would be the correct therapy. Four hundred thousand units of penicillin were given intramuscularly. At the end of five hours the swelling around the eyes had partly diminished and the patient was able to see. By the end of twenty-four hours the swelling of the left cheek had markedly decreased in size. The temperature remained normal. No change in respiration occurred nor did the patient complain of distress at any time. The patient received penicillin for five days. All swelling and crepitation had disappeared at the end of seven days (Fig. 2).

Historical Review

According to Edwards[3] the first report of subcutaneous emphysema of the head and neck following an interruption of the continuity of the mucous membrane of the mouth was that of Turnbull in 1870. Turnbull removed a bicuspid tooth from a bugler on a naval vessel. A short time later the bugler blew mess call, thereby causing an enormous swelling of the side of his face from which the tooth had been removed. In 1910 Parish[6] reported the occurrence of emphysema of the neck, cheeks and eyelids and of the skin over the ribs anteriorly in a twenty-eight-year-old male who had a tonsillectomy under an ether anesthetic. The patient left the operating room in good condition, but within a few minutes went into shock with marked facial emphysema, lividity of the face and cyanosis of the lips. The patient recovered uneventfully and in three days the emphysema had disappeared.

Later, von Hofe[9] reported three tonsillectomies

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in children complicated by emphysema of the head and neck. In each case, following the removal of the right tonsil, the right side of the face and neck swelled up with crepitation throughout the swollen area. The swelling and crepitation disappeared within five days in all cases. In each case the "ether hook" was suspended on the right side of the mouth and the air-ether mixture was blown by a pump against the right buccal membrane. Von Hofe believed that the most likely cause in these three cases was that air was forced into the subcutaneous tissue through the perforation of the tonsillar bed. He also stated that two other possibilities were perforation of an alveolus of the lung or entry of air through Wharton's duct.

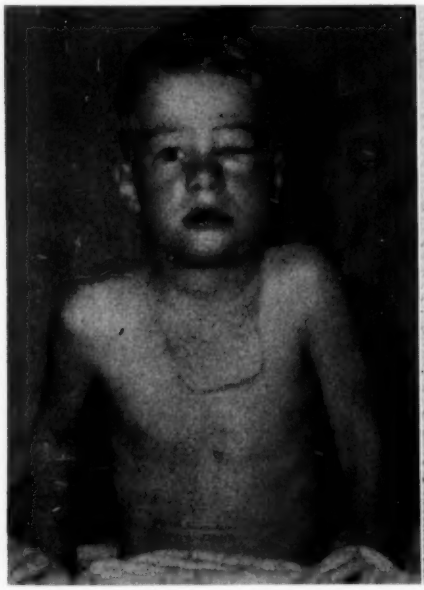

Fig. 1. Fifteen minutes after tonsillectomy and adenoidectomy left eyelids were swollen shut and marked swelling of the left cheek and neck was present. Crepitation was present in the swollen areas as well as in the area marked on the chest.

Fig. 2. The patient has a normal appearance seven days after appearance of subcutaneous emphysema.

Stevenson[7] also believed that air enters the subcutaneous space through the muscle fibers of the tonsillar beds. He reported a patient who developed constant bubbles in his saliva six hours after oxygen was blown through a tube onto the palate. Although subcutaneous emphysema was present from the forehead to the clavicles, it disappeared in five days without complications. Dickson[2] reported a case and stated that he also believed air passed directly through the tonsillar bed to the subcutaneous tissue.

Not all authors have accepted this explanation, however. Keen[4] reported subcutaneous emphysema over the left side of the chest, back and front, and over the lower part of the neck, which did not disappear for eleven days, in a five-year-old boy thirty hours after tonsillectomy and adenoidectomy. He believed the emphysema to be alveolar in origin. Thornton, Adams and Livingstone[8] reported a case of mediastinal and subcutaneous emphysema without pneumothorax following intratracheal insufflation anesthesia for craniotomy. No exhalation valve was present in the system. They believed that the intrabronchial pressure could have become elevated, causing a ruptured alveolus. Baker[1] recorded a case and stated that the complication was not particularly unusual, in spite of the few cases reported in the literature. He further stated that subcutaneous emphysema may occur during either general or local anesthesia and requires no special treatment. Further, the condition may be immediate or delayed, it occurs in both adults and in children, and with either the Sluder or dissection methods.

The etiology of the case Baker reported appeared to him to be a combination of a rupture of one or more alveoli in addition to seepage of air through the damaged muscle fibers of the tonsillar fossa.

Two of the most interesting case reports of subcutaneous emphysema following tonsillectomy are those of Mac Cready.[5] Tonsillectomy was done on a sixty-five-year-old male under local anesthesia with 1 per cent procaine. One hour after the patient had returned to his room a swelling suddenly appeared under the left eye following a severe attack of coughing. Crepitation was present over the left side of the neck and cheek. In forty-eight hours the swelling had disappeared. The second report was that of a forty-three-year-old woman who was given an ether anesthetic while a tonsillectomy was done by blunt dissection. At the end of the operation the patient was given a mixture of carbon dioxide and oxygen. The inhalation of these gases was prolonged and the patient clenched her teeth tightly. Suddenly there was a violent expiratory effort and a marked swelling appeared around the left eye. The eye was completely closed by the swelling of the eyelids and the left cheek was also swollen and crepitant. In forty-eight hours the swelling had disappeared.

Discussion

In our case, the most likely etiology is increased intrapharyngeal pressure due to a combination of coughing and vomiting and a tightly packed nose and nasopharynx, with consequent forcing of air through the tonsillar fossa into the subcutaneous tissues. The most persuasive argument for this explanation is that the emphysema did not occur until fifteen minutes after the patient left the operating room. Had a perforation been made in the larynx, or trachea during intubation, or had an alveolus ruptured because of high intrabronchial pressure, the emphysema would mostly likely have appeared during the operation as in von Hofe's cases. Furthermore, most of the emphysema was confined to one side of the face and more emphysema was present in the face than in the neck or chest, which suggests that the point of air entry was in the head rather than in the chest. It is conceivable, however, that a transient sticking of the expiratory valve could occur with a sudden rise in intrabronchial pressure and consequent alveolar rupture. Later, increased expiratory efforts could have forced air subcutaneously. Nevertheless, conditions for the production of subcutaneous emphysema were present here as in the other cases reported in the literature: tonsillectomy plus increased pressure within the pharynx. Although no objective data are available to substantiate it, reasoning suggests that the site of the etiology here is in the pharynx rather than in the trachea or lung.

As far as we have been able to determine from reading the literature no deaths have been reported due to subcutaneous emphysema following tonsillectomy. In our case the uneventful subsidence of the emphysema was again fortunate.

Summary

1. A report is given of a six-year-old boy who suddenly developed subcutaneous emphysema of the left eyelids and cheek, and of the anterior neck and chest following tonsillectomy and adenoidectomy. Conservative treatment resulted in the disappearance of the emphysema in seven days.

2. Ten other cases of subcutaneous emphysema following tonsillectomy are reviewed and summarized from the medical literature.

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RADIOPHOSPHORUS IN HEMOPOIETIC DISORDERS

CHARLES F. STROEBEL, M.D., DAVID G. HANLON, M.D.,
and PAUL W. SCANLON, M.D.
Rochester, Minnesota

USE OF radiophosphorus has passed beyond the experimental stage and now holds a significant place in the treatment of polycythemia vera and chronic leukemia. Its administration is becoming widespread as it is being made available to physicians who can satisfy the Atomic Energy Commission of their qualifications to handle it and give it to patients. Its use does not require expensive or elaborate equipment.

However, anyone who views the uncritical use of other agents, such as liver extract and vitamin B_{12}, in the treatment of hemopoietic diseases, cannot help but fear that radiophosphorus will be given when its use is not clearly indicated. Radiophosphorus yields ionizing radiation and can suppress the functions of bone marrow. Its use, therefore, must be founded on well-established diagnosis and definite indications.

Radiophosphorus is supplied as a solution of dibasic sodium phosphate that may be administered intravenously or orally. It yields beta radiation (electrons) only, in contrast to other radioisotopes which yield gamma rays in addition. The biologic effect of these electrons is similar to the secondary electrons excited by roentgen rays. When administered internally, it supplies total-body radiation.

The advantages in use of radiophosphorus lie in absence of radiation sickness, in ease of administration, in the short time required for treatment and in the simple, cheap equipment needed. The latter includes containers properly shielded to control the radiation, ordinary syringes and intravenous needles. Because radiophosphorus undergoes decay, the therapist must have a chart or slide rule to calculate the strength of the solution at the time of administration. The equipment must, of course, be used only for radiophosphorus. It is advisable to employ monitoring techniques to safeguard both those handling the material and the place where it is used from contamination. The therapist must know the fundamentals of radiobiology and safety precautions in radiation therapy and must have had experience in handling radioisotopes.

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The Mayo Foundation is a part of the Graduate School of the University of Minnesota.

Indications for Treatment

Polycythemia Vera.—The outstanding indication for the use of radiophosphorus is polycythemia vera, but the diagnosis must be clearly established. It is not sufficient to regard elevated values for hemoglobin, erythrocytes or cell volume per cent (hematocrit) as criteria. These are relative measurements, depending in part on the volume of plasma. A normal absolute erythrocytic volume with a decreased absolute plasma volume will give an increase in hemoglobin and erythrocytes and in the hematocrit value, a condition known as "relative polycythemia," or "pachyhemia" (thick blood). This is not an indication for radiophosphorus therapy, as erythropoiesis is normal.

The basic criterion for a diagnosis of polycythemia vera is an increase in the absolute erythrocytic volume as measured by one of the techniques for determining the volumes of whole blood, plasma and erythrocytes. These procedures include the dye methods (Congo red or Evans blue) or techniques using such radioisotopes as radiochromium, radiophosphorus or albumin labeled with radioiodine.

We use the dye technique, which can be performed in almost any laboratory. By this method, the normal absolute erythrocytic volume does not exceed 50 cc. per kilogram of body weight for adults of normal weight. Values between 50 and 60 cc. per kilogram are regarded as equivocal unless accompanied by substantiating evidence, such

as splenomegaly, leukocytosis, a leukemoid reaction or hyperplastic bone marrow. In the absence of one of these findings, the disease is best managed by phlebotomies and periodic observation until it is clear whether or not the erythrocytic volume is going to reach more definitive values. In our experience, erythrocytic volumes of 60 cc. or more per kilogram unequivocally indicate polycythemia.

It must be emphasized that the absolute erythrocytic volume is the essential issue, not the whole-blood volume, as the latter may be increased by an increase in absolute plasma volume. We have seen patients who have normal erythrocytic volumes but increased plasma volumes (up to 60 cc. per kilogram); this increase raises the whole-blood volume to more than 100 cc. per kilogram, which usually is conceded to be the upper limit of normal.

Important accessory information may be gained from study of smears of peripheral blood, in which regeneration of erythrocytes may be evident by increased polychromasia. Piling of erythrocytes is often seen but may be misleading if smears are not properly made.

Leukocytosis or myeloid immaturity or both may or may not be accompanying features. In only eighty-eight of a series of 241 cases of polycythemia vera studied by us was leukocytosis or myeloid immaturity noted. We have classified polycythemia vera on this basis: (1) the simple type, lacking leukocytosis or myeloid immaturity, (2) the type with an increase in mature leukocytes and (3) the leukemoid type, exhibiting myeloid immaturity. This classification is important as a gauge to prognosis and therapy. The leukemoid type tends to be more severe than the other types and presents a higher incidence of hematologic complications, such as the "anemia-leukemoid" state (severe anemia, pronounced splenomegaly and a leukemialike picture with regard to the leukocytes). The latter two types need larger doses of radiophosphorus on the average than does the simple type.

Studies of bone marrow may be of value, especially in the equivocal cases already mentioned. The marrow often presents a hyperplastic myeloid picture but it may not appear remarkable in some instances of the simple type. This should not deter treatment in the event of other unequivocal evidence of polycythemia vera. Studies of marrow are of especial value in advanced disease of the leukemoid type, in which an early change to myelofibrosis may be noted. In these studies it is imperative that units of marrow be obtained for embedding and sectioning so that the actual architecture may be seen.

Splenomegaly is an inconstant finding in the simple type but it is often present in the other two categories.

In some cases of uncomplicated asymptomatic simple polycythemia vera with absolute erythrocytic volumes of more than 60 cc. per kilogram, the process may be controlled by occasional phlebotomy, but care must be exercised to be certain that this method maintains hematocrit values of less than 55 per cent. Symptomatic improvement is not a reliable indication of control. If phlebotomy is needed oftener than every two months to produce objective control, we consider that use of radiophosphorus is indicated.

Patients who have polycythemia characterized by an increase in mature leukocytes or who have the leukemoid type should receive radiophosphorus after the hematocrit value has been decreased to near 55 per cent by phlebotomy.

Secondary Polycythemia.—An increase in absolute erythrocytic volume is present in secondary polycythemia but there is an evident cause, such as the anoxic anoxia of cyanotic cardiac disease or pulmonary impairment. If clinical differentiation is difficult, the presence of anoxia may be confirmed by studies of the oxygen saturation of arterial blood and measurements of pulmonary function. Secondary polycythemia is common in persons living at extremely high elevations.

Certain brain tumors, such as cerebellar hemangiomas, may produce absolute polycythemia. Other brain tumors may be associated with relative polycythemia due to low plasma volume. True polycythemia occasionally is seen with hypernephroma; it may disappear with removal of the tumor, recurring if metastasis appears.

Chronic Myelocytic Leukemia. — Treatment with radiophosphorus can achieve results comparable to those seen after roentgen therapy in early chronic myelocytic leukemia. It is also effective in more advanced disease, although pronounced splenomegaly may make focal roentgen therapy to the spleen preferable. When the disease is in its late stages and is refractory to roentgen therapy, radiophosphorus is seldom effective.

It is important to recall that not all instances of leukocytosis, leukemoid reaction and splenomegaly represent chronic leukemia. Severe sepsis, especially that due to pneumococci, may increase the number of leukocytes to more than 100,000 per cubic millimeter. Diffuse granulomatous disease, complicated lymphoma and malignant tumors with infection and necrosis may produce leukemoid pictures. When the diagnosis is not certain, it is better to defer use of radiophosphorus, which is not an agent for emergency treatment. Its effects are not immediate enough to be of benefit in critical situations. Usually the insidious onset, pronounced myeloid leukocytosis and splenomegaly leave little doubt as to the diagnosis of chronic myelocytic leukemia but, as a rule of thumb, it is sound practice to observe patients who have myeloid leukocytosis for several months until the diagnosis is certain.

It is by no means clear when to start treatment in early, mild chronic myelocytic leukemia without anemia or symptoms. It is not established that early treatment in such cases will prolong life. The specter of acute leukemia in persons who handle ionizing materials for radiation therapy cannot be entirely disregarded. It is well to have a definite reason for therapy, such as anemia, fever, undue exhaustion, advancing splenomegaly or lymphadenopathy. The same principle underlies use of roentgen therapy.

Studies of bone marrow in early chronic myelocytic leukemia reveal a hyperplastic myeloid marrow that is not pathognomonic. Such studies are of particular value if there is a question of acute leukemia and in long-standing conditions in which myelofibrosis may be present.

Patients sometimes exhibit a large, firm spleen, normal erythrocytic values or possibly anemia, a myeloid leukemoid reaction in the peripheral blood and a hyperplastic myeloid bone marrow. Often the leukocytes number less than 75,000 per cubic millimeter. Normoblasts and abnormally shaped erythrocytes are present in the peripheral blood. This may be the picture in advanced chronic myelocytic leukemia, a late sequela in polycythemia vera, or it may appear without a definite history other than that of a slowly progressive splenomegaly.

The nature of the condition just described is obscure, although it is sometimes called "atypical myelocytic leukemia." Many of these patients have a life duration longer than that observed in most patients who have chronic leukemia. Perhaps the condition should be called the "leukemialike syndrome" or the "anemia-leukemoid state."

This syndrome is pointed out simply to distinguish it from typical leukemia and to note that it is usually resistant to treatment with radiophosphorus. The usual symptom of which the patient complains is abdominal discomfort, pain or pressure from the large spleen; radiophosphorus will not reduce the size of the spleen, although focal roentgen therapy may be of temporary benefit.

In similar cases in which actual replacement of bone marrow with fibrous tissue is observed, total-body suppressive therapy or any type of radiation therapy is frowned on for fear of destroying regions of ectopic myelopoiesis in the liver, spleen and other sites. However, if there is an established hemolytic component, focal roentgen therapy or splenectomy occasionally is of benefit.

Chronic Lymphocytic Leukemia.—Radiophosphorus may be used in the treatment of chronic lymphocytic leukemia, although roentgen therapy or use of nitrogen mustard and related chemicals appears to be better. Radiophosphorus or total-body roentgen therapy, however, has a definite place in the management of widespread disease and when poor response is shown to focal roentgen therapy in the early stages of disease. Radiophosphorus is seldom helpful in late stages when the disease is resistant to roentgen therapy. Nitrogen mustard may prove of benefit in this situation.

Conditions in which Radiophosphorus is of Questionable or Doubtful Value

Multiple Myeloma.—It was hoped that radiophosphorus would be of benefit in multiple myeloma, but total-body radiation with radiophosphorus or roentgen rays has proved disappointing, although a rare patient has undergone symptomatic benefit. Use of urethan, cortisone or focal roentgen therapy, although of limited value, is preferable. Focal roentgen therapy directed, when possible, to painful lesions may afford palliative and symptomatic relief.

Lymphoblastoma.—Patients who have any of the various lymphoblastomas have not experienced effective response to radiophosphorus therapy.

Better results are obtained with roentgen therapy and nitrogen mustards and related chemicals.

Osseous Metastasis.—An occasional patient who has metastatic carcinoma in bones will experience relief of pain after treatment with radiophosphorus but results have been disappointing. Use of hormones and focal roentgen therapy is preferable for osseous metastasis from carcinoma of the breast or prostate. Melanomas are as resistant to use of radiophosphorus as they are to roentgen therapy.

Thrombyocytosis.—An increase in the number of platelets may occur in polycythemia vera (15 per cent of cases) or may be a prominent feature in early chronic myelocytic leukemia. The response of the platelet count to radiation therapy parallels improvement in the blood picture. In general, radiophosphorus therapy has not been satisfactory in controlling the high platelet counts seen in postsplenectomy states, in megakaryocytic hyperplasia and in thrombocytosis of undetermined origin.

Attempts to decrease normal platelet counts in chronic thrombosing disease have not been successful within safe limits; the depression of platelets is too brief to be worth while. Anticoagulant therapy is preferable.

Contraindications to Radiophosphorus Therapy

Acute Leukemia.—Usually no change in cellular type is obtained by use of radiophosphorus in this disease. As with other types of radiation in acute leukemia associated with pronounced leukocytosis, either no response to therapy is noted or a pronounced and rapid decrease in the number of leukocytes occurs. No clinical improvement ensues from use of radiophosphorus in acute leukemia.

Leukocytosis Not Due to Polycythemia Vera or Chronic Leukemia.—There is no point in correcting leukocytosis not due to the two diseases just named. The leukocytic increase is often part of a defense mechanism.

Hypersplenic Syndromes.—In hypersplenic syndromes, total-body radiation with radiophosphorus or roentgen rays is not sufficiently concentrated in the spleen to be of value. It may even depress the hyperfunctioning bone marrow, which is trying to maintain normal cellular values in the face of abnormal immunologic mechanisms. Control of hypersplenism needs more drastic focal management, such as splenectomy, focal roentgen therapy or use of steroids.

Relative Polycythemia.—This is not a disease of hemopoietic organs but a deficiency of plasma volume; it is not satisfactorily affected by radiation.

Hypoplasia of Bone Marrow.—Any condition associated with a hypoplastic state of the bone marrow contraindicates use of radiophosphorus.

Dosage Schedules for Radiophosphorus

Polycythemia Vera.—Preliminary phlebotomy is used to reduce the immediate risk of vascular complications, such as thrombosis. One or two phlebotomies, with removal of 500 cc. of blood on each occasion, are usually sufficient unless the erythrocytic volume is extremely great. As already indicated, an attempt is made to decrease the hematocrit value to 55 per cent before administration of radiophosphorus is begun.

After phlebotomy, the patient is given a single dose of radiophosphorus either intravenously or orally. When the oral route is employed, the dose must be approximately 30 per cent more than is given intravenously to compensate for failure to absorb the entire amount. Doses to be presented are those for intravenous administration.

In the past few years we have tended to give smaller amounts than were used initially, preferring to repeat the dose in eight weeks rather than give unnecessarily large doses at first. A study of the amounts of radiophosphorus needed to produce remission for at least one year showed that the range of dosage in treatment of the simple type of polycythemia was 3 to 21 millicuries (divided dosage). The same ranges were noted for treatment of the type associated with increased numbers of mature leukocytes and the leukemoid type; however, the mean effective dose for the simple type was 5 millicuries, whereas it was 7 to 8 millicuries for the other two types.

We usually administer 4 millicuries to patients who have the simple type of polycythemia; patients who have the other types receive 5 millicuries.

The patient is again studied in eight weeks with regard to hemoglobin, hematocrit, erythrocytes,

leukocytes and platelets. If the values are normal, further treatment is deferred. Studies are repeated at intervals of eight to twelve weeks, and treatment is reinstituted when relapse occurs.

Secondary Polycythemia.—In many instances, treatment of the primary disease, with occasional phlebotomy, will improve the polycythemic state so that suppression of bone marrow is not needed. In extremely rare cases in which these measures are insufficient, radiophosphorus is administered as it is in polycythemia vera, although the effects are not so certain.

Chronic Leukemia.—Extended treatment is needed in chronic leukemia. In former years, we administered radiophosphorus twice weekly until the number of leukocytes decreased to 40,000 per cubic millimeter. At this point treatment was discontinued; the patient returned for more radiophosphorus when symptoms reappeared or if pronounced leukocytosis recurred.

During the past year we have used a modification of the Osgood technique.[1,2] The patient who has chronic myelocytic leukemia is given 40 microcuries of radiophosphorus per kilogram of body weight. This amount is given at intervals of two weeks until the leukocyte count decreases to less than 30,000 per cubic millimeter. Observation is then continued at biweekly intervals and treatment started again as soon as the number of leukocytes begins to increase.

It is stated that this method affords greater longevity, efficiency and comfort than do other methods.[1,2] We have not used this technique long enough to judge the relative effects on duration of disease, but we have been impressed with the fact that less radiation (9 to 15 millicuries) is used than in the older method (20 to 25 millicuries). As noted previously, later stages of the disease may require more therapy or be entirely resistant.

Complications of Treatment

Because radiophosphorus is administered as dibasic sodium phosphate, in the form of ions that are common in the body, and because radiophosphate is identical chemically with natural phosphate, no chemical intoxication occurs. The danger lies in the potentiality of radiophosphorus depressing the function of bone marrow too far. As the use of radiophosphorus has been carefully controlled to date, instances of serious hypoplasia of the marrow are extremely few, but this remains a hazard if radiophosphorus is not used cautiously.

Acute leukemia that appears terminally in some cases of polycythemia and of chronic leukemia has been laid at the door of radiation therapy but this relationship is by no means clear. Death from "hematologic complications" occurred in 13 per cent of a series of cases of polycythemia in which radiophosphorus was not given.[3] However, adequate data were not at hand to indicate if death was due to acute leukemia or to one of the "leukemialike" states. As already noted, radiation may be a cause of acute leukemia, as evidenced by the abnormal incidence of leukemia in workers handling ionizing radiation. Even if it is assumed that acute leukemia is a risk of such treatment, this risk is much smaller than the possibility of vascular occlusion and hemorrhage so commonly seen in days before radiophosphorus therapy.

About 10 per cent of deaths in patients who have polycythemia vera and who receive radiophosphorus occur as the result of "leukemialike" states, the "anemia-leukemoid state," "atypical chronic myelocytic leukemia" or myelofibrosis with myeloid metaplasia. However, the anemia-leukemoid state occurs in patients who have had no radiation or extremely small amounts. It is suspected that the "anemia-leukemoid state" may be a late sequela of myeloproliferative diseases, if life is not terminated earlier by some other misadventure.

In polycythemia vera, the average age at death in our series of patients receiving radiophosphorus was 64 years, while the average age at death was 54 years in patients treated by other means. This, along with general clinical impressions, indicates that carefully administered radiophosphorus does not pose serious complications of treatment.

Summary and Conclusions

Use of radiophosphorus is established therapy for polycythemia vera and for chronic leukemia of the myelocytic or lymphocytic type.

Use of this radioactive agent is contraindicated in acute leukemia, leukocytosis not due to polycythemia vera or chronic leukemia, hypersplenism, relative polycythemia and all types of hypoplasia of bone marrow.

Administration of smaller doses than those used formerly is suggested.

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Laboratory Aids to Medical Practice

Sponsored by
The Minnesota Society of Clinical Pathologists

CAREER: MEDICAL TECHNOLOGY

GEORGE G. STILWELL, M.D.
Rochester, Minnesota

Nature is supposed to detest a vacuum; when such a situation exists, nature rushes in, either mechanistically or teleologically, in an attempt to fill the vacancy. A void, albeit partial in extent, exists today in the field of medical technology. Whether it be the fault of nature or some other agent, there appears to be no concerted rush to fill this partial vacuum. The material needed for stuffing this particular void is young people of either sex, although at present 92 per cent of registered medical technologists are women.

Why is the Shortage of Medical Technologists Important?

The internist, the surgeon and the general practitioner, as well as the pathologist, should be concerned about the inadequate supply of trained medical technologists. All physicians feel the impact of this paucity because the shortage of qualified technologists is reflected in lessened efficiency in the application of laboratory medicine to diagnosis, prognosis and treatment. The importance of the relationship between clinicians and pathologists goes without saying, yet the potential benefits of this relationship cannot be realized optimally if the pathologist is handicapped by lack of assistants in the laboratory. The medical technologist is as important in the total practice of medicine as is the nurse, and shortages of personnel in either of these professions cannot help but affect the practice of medicine unfavorably.

What Is Medical Technology?

Medical technology is one of the more recent adjuncts to the scientific and modern practice of medicine. The work of the medical technologist is an integral part of the practice of clinical pathology, and the pathologist would be lost without the indispensable aid of these handmaidens in the laboratory diagnosis of disease.

The medical technologist starts with an interest in science and the possession of a considerable degree of manual dexterity. Accuracy, calmness under pressure, efficiency and cheerfulness are primary requisites. The medical technologist who has attained certification by the Registry of Medical Technologists of the American Society of Clinical Pathologists has had a minimum of two years of collegiate work plus twelve months' training in an approved school of medical technology. Many spend three years in college and then obtain a Bachelor of Science degree on completion of their technical training in the approved school. The certified medical technologist must then pass a nationally conducted examination.

The great majority of medical technologists work in hospital laboratories performing the multitude of important laboratory tests with which all physicians are familiar. Others work in clinics, physicians' offices, private laboratories or research institutions. The work of the medical technologist is satisfying, the demand for such persons is great and the compensation is good.

What is an Approved School of Medical Technology?

Many physicians who are consulted by young people seeking advice about entering the field of medical technology enthusiastically recommend the profession but may be a bit hazy about exact directions for correctly steering such persons to the proper place. Any qualified pathologist can give advice and information concerning acceptable schools for medical technology.

An approved school of medical technology is

From the Section of Publications, Mayo Clinic, Rochester, Minnesota.

This is the twenty-second in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between clinicians and pathologists.

one that has been inspected and accepted by the Council on Medical Education and Hospitals of the American Medical Association. All training of technologists in such schools must be done under competent medical control. Each school must have an adequate and qualified teaching staff. The director of the school must be a graduate in medicine who is certified in clinical pathology by the American Board of Pathology or who has had training and experience in clinical pathology acceptable to the Council. Approximately 500 such schools exist in the United States; a complete list can be obtained from the Council on Medical Education and Hospitals of the American Medical Association, 535 North Dearborn Street, Chicago 10, Illinois.

What is an Unapproved School?

A number of commercially sponsored schools allegedly for the purpose of training "technologists" are found throughout the United States, including Minnesota. These schools offer to graduates of high schools a short course, usually ranging from 9 to 12 months, at a relatively high tuition. Such schools are not on the list approved by the American Medical Association because the instruction in these unapproved schools is given by nonprofessional persons, the curriculums are inadequate and substandard, and these schools have no hospital affiliations. The students accepted by these schools are, of course, unqualified according to the standards set up for approved schools.

Many students who "graduate" from these commercial schools that have no hospital connections are amazed and disappointed to find on applying for work that their training and knowledge are completely inadequate to qualify them for work in many laboratories. The undesirability of recommending these unapproved schools to young people interested in a career in medical technology is obvious. Again, the pathologist can advise a physician who is in doubt about which schools are approved and which are substandard.

What is Being Done About the Shortage of Medical Technologists?

Because the afore-mentioned vacuum has not been adequately taken care of by natural forces, some assistance is being offered by several groups interested in the field of medical technology. A National Committee for Careers in Medical Technology has been formed by three sponsoring organizations, namely the American Society of Medical Technologists, the American Society of Clinical Pathologists and the College of American Pathologists. These groups have co-operated in the establishment of an active program of recruitment to inform young people of the satisfactions and opportunities offered by a career in medical technology.

The most tangible evidence of activity in this program is in the form of a film entitled "Career: Medical Technology." This is a 16-mm. movie in color and sound, with a running time of 24 minutes. It is a professional job turned out at a cost of many thousands of dollars and a film whose quality is such that pathologists and medical technologists can be proud to present it anywhere.

The primary purpose of this film is to tell the story of medical technology to high-school and college students and to high-school counselors and other school personnel to whom students turn for advice concerning their future careers. It is suitable for showing during the Career Day held by many high schools in Minnesota. It can be used as a television educational feature. It can be shown before science classes and clubs; it can be presented before high-school faculties so that the teachers can know more about the profession of medical technology.

This film will attain its greatest usefulness when its showing is accompanied by the presence of both a pathologist and a medical technologist; they can set the scene and, as a postlude, answer questions raised by the film.

The national première of this film was held in Washington, in September, when it was enthusiastically received at the annual meeting of the American Society of Clinical Pathologists. The first Minnesota showing was held in November. Prints of this film have been purchased by both the Minnesota Society of Clinical Pathologists and the Minnesota Society of Medical Technologists; these prints are available without charge for showing to interested groups. Inquiries regarding use of these films may be directed to Dr. Kano Ikeda, Charles T. Miller Hospital, St. Paul, who is chairman of the Committee on Medical Technology of the Minnesota Society of Clinical Pathologists, or to Virginia Burris, M.T. (ASCP), 57 Inner Drive, St. Paul, who is president of the Minnesota Society of Medical Technologists.

(Continued on Page 907)

President's Letter

THE GREATEST GIFT

"It doesn't seem like Christmas." How many times have you heard someone say that around this time of the year?

But why shouldn't it seem like Christmas? Is it because we have commercialized this sacred holiday to such an extent that we leave out the real reason for our celebration? Or is it because we are so wrapped up in our own mundane affairs that the holiday is upon us before we have had time to prepare for it mentally and spiritually?

The birthday of Christ should find us, as physicians, keenly attuned to the meaning of healing. We are engaged in the repair of bodies, and the prevention of damage to them. Christians are likewise keenly attuned to the meaning of healing—but to the healing of souls instead. Therein lies a sound kinship of the two. For a deep abiding faith in God is a tremendous asset to the healing of the body, and Medicine knows the value of such faith.

A strong thread of such faith should run through the hearts of men of medicine in their everyday practices. Because we are mere men, we are eternally dependent upon a higher power, and without it, we should be lost.

At Christmas we develop a greater regard for our fellow men; we nourish in ourselves that latent love for all people which has lain dormant in us throughout the year.

But if it doesn't seem like Christmas, whose fault is it? It is ours. We are to blame if we feel that the holiday is merely something for which we have to prepare. We should not have to cultivate the Christmas spirit—the spirit of giving, patterned after the greatest Gift of all, should be an integral part of our daily living and working. It should be automatic—a feeling that gives us in return great contentment, sincere good will, and infinite peace.

In this, my last letter to you during my term as president, I would like to tell you that I have found countless instances of this fine spirit throughout the year. The men with whom I have had the privilege to work during the past twelve months, have proved to me that this spirit is not always dormant. I have been gratified to have been associated with men of this high caliber. My deep thanks and appreciation to all those who have made my term a satisfying and rewarding one.

I would like to leave this parting thought with you: Because we are members of a great profession, let us at all times sincerely try to be worthy of the respect and reverence accorded us by our patients, friends and colleagues. We can do no better than to imitate the life, the faith, and the indomitable spirit of the man whose birthday we are celebrating now. Let us constantly strive to "make it seem like Christmas."

Best wishes for a blessed holiday season.

President, Minnesota State Medical Association

Editorial

Arthur H. Wells, M.D., *Editor;* Henry G. Moehring, M.D., and John F. Briggs, M.D.

OUR MOST POTENT WEAPON AGAINST TUBERCULOSIS

THERE is nothing so important in controlling a disease as a specific test to determine its presence while it is microscopical in size as well as during later stages of its development. Everywhere physicians lament the fact that a test is not available to detect the presence of cancer before the lesions are large enough to be located by other methods of examination. If such a test were available, the organs of the reactors which cancer frequents could be examined often, and in all probability many in the silent stage would be located before they have metastasized.

In tuberculosis, such a test is available in the tuberculin reaction. It is well known that within an hour after invasion with tubercle bacilli occurs, lesions have begun to form. Within seven or eight weeks, the tissues of the host, the skin included, are so sensitized to tuberculo-protein as to react characteristically to tuberculin. Therefore, at this time a tuberculin reaction indicates the presence of lesions containing tubercle bacilli. The sensitivity of tissues to tuberculo-protein apparently is maintained as long as living tubercle bacilli reside in the body.

When the tissues first become sufficiently allergic to react to the tuberculin test, lesions are so small that they can rarely be located by any type of examination, including roentgenogram inspection. However, such lesions may subsequently develop to clinical proportions. In fact, all clinical lesions begin in this way.

The tuberculin test provides so much more information about tuberculosis than any other part of the examination that it should be routine with every physician, regardless of where he works or what phase of medicine he does.

Some of the values of the tuberculin test are:

1. It detects the presence of tuberculosis in the human body much earlier than any other phase of examination.

2. Since tuberculosis is a contagious disease, the physician knows that the tuberculin reactor has been in contact with someone who is eliminating tubercle bacilli. Therefore, examination of the reactor's associates often brings to light the person who is responsible for transmitting the infection. Thus, the tuberculin test is our best epidemio-logical measure.

3. The only accurate method of determining with reasonable promptness the effectiveness of a tuberculosis control program in a community or a larger area consists in determining whether the incidence of tuberculous infection among children is decreasing from time to time; that is, whether fewer babies and young children are infected now than were those in the same age period a few years ago. This can be determined only by testing with tuberculin.

4. There is now some reason to believe that antimicrobial drugs, plus the body's defense mechanism, may destroy all tubercle bacilli if administered soon after the invasion occurs while lesions are still small and vascular. The only way to determine when this condition is present is to test nonreactors periodically to find those promptly after they convert to reactors. It now seems more than likely that this will be the ideal treatment of the future. If so, periodic tuberculin testing will be indispensable. Therefore, the tuberculin reaction will be the only method by which the physician will know when to start treatment. In fact, such treatment of recent converters is already being employed by a number of physicians.

5. It is believed that when all tubercle bacilli are destroyed in primary lesions, sensitivity of the tissues will disappear, and, therefore, the individual will cease to react to tuberculin which will be the indication to stop treatment.

6. Whenever tuberculosis is regarded as a compensable disease, much injustice may result unless periodic tuberculin testing is done among all concerned. For example, a lesion demonstrated by the x-ray shadow it casts, wherever a person is employed at the time, may be the result of an infection acquired months or years before in a different place. Thus, the liability dates to the times when the infection was allowed to occur.

Such accurate information is obtainable only by periodic tuberculin testing.

In the State of Minnesota there is now evidence that only slightly more than 20 per cent of the citizenry harbors tubercle bacilli and therefore reacts to tuberculin. Actual testing indicates that most of the persons now harboring tubercle bacilli are in the older age bracket. Among seniors in high school rarely are more than 4 to 8 per cent infected, and among grade school children 1 to 2 per cent in rural areas and 3 to 4 per cent in cities. There are many grade schools in which no child has been infected.

As the incidence of tuberculous infection decreases, the value of the tuberculin test increases.

A diagnosis of clinical tuberculosis is never justified in the absence of a tuberculin reaction or the recovery of tubercle bacilli. Practically everyone who is eliminating tubercle bacilli reacts to tuberculin; hence, the importance of this test in diagnosis.

In Minnesota, the tuberculin test is by far the most potent weapon the members of the State Medical Association have in their statewide tuberculosis eradication program.

J. ARTHUR MYERS, M.D.

HELL HATH NO FURY

WE have read that hell hath no fury like a dues-paying professional society member grown scornful of his society's publication. Well, why shouldn't he be furious? Perhaps his reasons for becoming scornful are invalid to anyone except himself. Nevertheless, his scorn and fury should be heard. The society's publication is for the society members, not just because they pay for it, but because they are the sole excuse for the existence of the publication. If the publication strays from serving *one* member, no great furor arises, but if one member, conscientious enough to read his society's publication, is aroused to fury, the likelihood is that other members are similarly aroused and that the cause of the discontent merits attention.

Andrew Jackson is credited with the observation that one determined man constitutes a majority. Times and the sense of what is fitting have changed since Old Hickory's day, but we still listen with profit to the voice of the minority in any group, whether it be a hospital staff or the nation. Witness our unwillingness to abridge the Bill of Rights because we think that any probable benefit to society would be outweighed by the chance of injustice to a citizen. The minority is at once a goad and a brake to those who determine the course of society and to society itself . . . a necessary and wholesome goad or brake. It is on the activity of pressure groups that democracy depends for its continuance as a democracy, for how is the voice of the people to enter government if unheard? The summation of the intensities and directions of these forces from pressure groups, of whatever size, establishes the course of a democratic society. Thus, even the negative forces, the brakes, make their influence felt.

So much for generalities. Now we would point out that the editorial board of MINNESOTA MEDICINE is not soliciting furious criticism from any State Medical Association member grown scornful, but that the board is open to suggestions and comments from dues-paying members and, for that matter, from anyone interested enough to bestir himself to make comments and criticisms about MINNESOTA MEDICINE. The editorial board is not able to guarantee an unimpassioned and totally objective appraisal and reaction to all criticism, but it will respond to such criticism just as certainly as any executive body of a democratic society responds to forces from within that society. As further assurance that any constructive criticism you have to offer will meet an interested audience, the editorial board declares itself unanimously in agreement with Aristotle's dictum that it is the discriminating eater and not the cook who is the best judge of the dinner. So let us have your contributions, be they critical, scientific, or otherwise.

H. G. M.

WHY THIS LIVELY INTEREST IN COMMON STOCKS?

THE period of speculative excesses of the late 1920s was followed by a long period of liquidation. During the thirties, we were in a depression during which much reliance was placed upon security guaranteed by a benevolent government. We witnessed a period of rising government debt, rising prices, and the beginning of inflation. Then

followed an expensive war with a huge increase in government debt. Savings were almost exclusively employed in the purchase of government bonds, insurance, annuities, and similar forms of guaranteed dollar contracts. In the years following the war, the holders of matured government bonds found that inflation had done something to their dollar in terms of purchasing power. Owners of pure dollar contracts had suffered a loss in purchasing power of some 40 per cent. Confidence in government paternalism faded as people realized that everything the government had to give must somewhere be taken from the people. The realization grew that the only source of wealth stemmed from the production of goods and services which is the function of business. Since ownership of business or the ownership of common stocks are synonomous, all financial security, in the long run, must stem from common stocks.

A large mutual fund released some observations which seemed startling to the public. This fund revealed recently that all who had invested $500 per month, plus quarterly dividends, for any twelve-year period of the fund's existence, were in a position to take income of $500 per month without disturbing the principal amount. Upon further examination of the results of other leading mutual funds, it was found that this remarkable performance was common. It follows very closely the pattern of the one-hundred-year history of common stocks as reported by the Cowles Commission, which shows an average growth of 3 per cent and income of 6 per cent annually.

A person may inquire, "What is responsible for such a superior record of growth and income, and what are the prospects for its continuation?" First, the outstanding characteristic of this country is growth. Since all material progress in this country is dependent on savers exchanging accumulated capital for equities, these equities or common stocks are the means through which wealth is created. It follows, therefore, that our government bonds are good because common stocks are good.

Despite periods of interruption, the long-term growth in value of common stocks parallels the growth of our economy. Seven thousand new mouths must be fed each day. Americans constantly demand higher living standards. Industry strives to provide these needs through research. Expenditures for research and new product development will be stepped up even higher as business competes for a share of the expanding consumer market. New industries such as electronics and nucleonics will contribute substantially to economic progress. An insight into what the future holds may be had from the remarks of Philip Reed, chairman of General Electric Company, who cited reasons why General Electric anticipates producing in the next ten years as much electrical equipment as they have produced in the previous seventy-five years.

The greatest factor in the market and the hardest to evaluate is the institutional buying of common stocks for income and participation in our expanding economy. Insurance companies, pension funds, trust companies and even savings banks in New York are turning to common stocks for better yields. It is the combined strength of these long-term investment-minded institutions, coupled with a new look in government fiscal policies, which has helped to reawaken investors' interest in common stock investing.

NICHOLAS V. SCHAPS
J. M. Dain & Company
Investment Securities

POISONS, A KILLER IN CHILDHOOD

YOUR attention is directed to the article in this issue on "Poisons, a Killer of Children in Minnesota." Putting a padlock on the medicine cabinet is not the complete answer. We must remind our families with small children that aspirin may kill. Home safety check lists include dangers under the kitchen sink, around supplies of insecticides and rodenticides, associated with fuel tanks, cleaning solutions, et cetera.

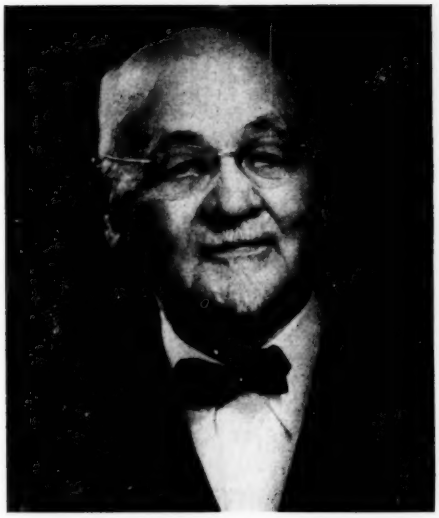
Morbidity from poisons, thank goodness, far exceeds mortality. Physicians have the moral responsibility of preventing these hazards as conscientiously as giving innoculations for diphtheria and whooping cough.

Your serious consideration is directed to the contributor's comments on a poison control center for Minnesota comparable to what has been set up in Philadelphia and Chicago.

TAGUE CHISHOLM, M.D.

E. E. Novak

Minnesota's "Physician of the Year"



Dr. E. E. Novak, New Prague, has been given Minnesota's "Physician of the Year" award by the Minnesota State Medical Association. The eighty-one-year-old doctor has practiced in New Prague for fifty-nine years, and is still active in his daily practice.

Dr. Novak came to New Prague in 1895 when the town was just beginning to look like a town. It was still frontier land, rugged and growing, and most of it had to be hewn out of dense timber to make the small farms of the area.

During the fifty-nine years of his practice in New Prague, Dr. Novak has grown with the town. From a struggling physician, he has come to be one of the most respected men of the community, revered and loved by all for his strong character, sincere good will and devotion to his people.

Besides practicing general medicine twenty-four hours a day all these years, Dr. Novak has found time to be of help to his community in many other fields: agriculture, education, cattle and horse breeding, civic affairs, financing, business and sanitation. In agriculture, he was a leader in promoting use of scientific methods, such as crop rotation, and better use of crops and farm animals. When he first arrived, the farmers were raising small crops, mostly wheat, and were milking a few scrubby cattle that weren't really dairy cattle. Dr. Novak, having come from a farm in Iowa, began introducing progressive methods to his neighbors. Many a farmer laughed at his "new-fangled" notions when he planted alfalfa, began raising tuberculosis- and brucellosis-free herds, shipped to market the first carload of hogs in the county and introduced diversified farming procedures. He helped organize the co-operative creamery of New Prague after realizing the need of the farmers in the area.

In the field of education, Dr. Novak was also very active. He served on the local school board for forty years, was active in the PTA, was president of the Minnesota State School Board association and received its distinguished service award. At present he is a Regent of the University of Minnesota, and as such has served admirably, with an avid interest in the medical school, the agriculture department, and all other fields as well.

His influence in the business and financial affairs of the town has been valuable over the years. He was one of the founders of the First National Bank of New Prague in 1903, and helped the State Bank of New Prague over the depression in the difficult years. His influence was not primarily financial, but was based on the confidence, trust and integrity of his character and the high regard the people have for him. Dr. Novak was elected as mayor of the town for two terms and presented sanitation projects and progressive ideas which did much to improve the growing community.

Dr. Novak has always been interested in breeding pure bred cattle and horses. Several of his

cattle have won coveted national honors. The doctor has been an active and honored member of the Red Poll Cattle Club of America, serving as its president for many years, and receiving its distinguished service award. He helped found the Southern Minnesota Livestock Show and has been a moving force in its growth.

There are countless stories that can be told about the doctor's struggle to practice good medicine throughout the severe early days around the area of New Prague. Poor communication, inadequate roads, reluctance of people to submit to new successful treatments, poor medical facilities, all combined to make his practice a hazardous one, especially when the winter months brought deep snow and afforded difficult travel, at the best, to many of his scattered patients. He used a bicycle, horses, wagons, buggies, and had the first car in town to get to his patients; often he went by foot to administer to the sick.

Although his honors are many, his greatness is reflected best in the firm respect that the townspeople have for him. After he had practiced fine medicine for fifty years in the area, the town and surrounding area gave him a gala tribute in a testimonial banquet and program—and the crowd was overwhelming. Tribute was paid to all his fine deeds by friends and neighbors, many of whom he had treated as children and adults, and many of whom he had brought into the world.

Many exalted words have been said of the good doctor—all of them deserving and accurate. But the greatest tribute paid to him is the love and affection of his fellow citizens. What the people feel about their doctor has been said in many ways—in an old man's crying because the doctor had saved his wife's life after the man had complained about the $7 charge; in a young child's relief from the pain of a broken leg or arm; in the grateful tears of an entire family when he answered a desperate call in the middle of the night; in the great fun and competition he has enjoyed in racing his own purebred horses; in the conflict and sense of something well done, experienced when he won one of his many battles against obstacles such as stubbornness and obstinacy standing in the way of medical progress; in the secure knowledge that a life has been, and still is, being devoted fully to the noble service of his neighbors, his community, his state and his nation.

RADIOPHOSPHORUS IN HEMOPOIETIC DISORDERS

(Continued from Page 886)

Radiophosphorus does not pose a chemical hazard but does present a radiation hazard if care is not taken in selection of cases and administration of the material.

The etiologic relationship between radiophosphorus therapy and acute leukemia is not established but remains a possibility. However, if this relationship is assumed, it is a small risk in view of the reduced incidence of vascular catastrophes in patients treated with radiophosphorus.

The relationship between "anemia-leukemoid states" and use of radiophosphorus is not established, but occurrence of these states without antecedent radiation therapy suggests they are sequelae or late phases of some myeloproliferative disorder.

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Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

NINE MILLION NOW COVERED BY INDEPENDENT HEALTH PLANS

A new government report shows that 9 million persons carried some protection under independent health plans at the end of 1953. This figure includes 6.9 million for hospitalization, 6.6 million for surgical benefits, 5.4 million for medical benefits in office, home or hospital. Also, 3.4 million were eligible for diagnostic services and 600,000 for dental care.

These figures are quoted in a recent issue of the *"Washington Report on the Medical Sciences."*

Unions First in Growth

One of the most striking findings was that in the past five years union plans have gone from eighteen in number, with less than 500,000 enrollment, to seventy-six having a membership of 2.8 million.

The report is based on a survey which covered 299 independent plans—consumer-controlled cooperatives, privately operated prepayment clinics, plans conducted by medical societies and fraternal groups, and other types unassociated with insurance companies, Blue Cross or Blue Shield.

The study was made by the Division of Research and Statistics, Social Security Administration, and the figures presented are from a preliminary report.

Plans Varied

Another important point is the great diversification of the plans studied. Some use community doctors, the report states, others follow the panel system and many give direct services in group service clinics. Although none are mentioned by name in the report, it may be disclosed that the study included such nationally known independent groups as Health Insurance Plan of Greater New York (HIP), the Kaiser system on the West coast, Ross-Loos Clinic in Los Angeles and the health plan sponsored by United Mine Workers. A total of 132 plans, covering 2.9 million persons, provide benefits through some form of group medical practice, the report says.

The Washington report notes that the 9 million beneficiaries are divided almost equally between employes and dependents. It continues:

"That the percentage of dependents is not higher is ascribed to the fact that employes alone are eligible for prepaid benefits in 40 per cent of the plans. Membership ranges from less than 100 in one co-operative to 1.4 million (workers and dependents) in the largest plan covered in the survey. Surprisingly, Connecticut ranks No. 1 among the states for hospitalization coverage in independent plans (1,110,000), the explanation being that it includes enrollment in a former Blue Cross plan."

Enrollment by States

The report further breaks down the enrollment in these plans by states: New York—1,496,000; Connecticut—1,114,000; Ohio—978,000; California—861,000; Pennsylvania—691,000; Washington—546,000; West Virginia—439,000; and Michigan—425,000.

Developments Cited

Besides rapid expansion of union-sponsored health plans, the survey cites certain developments in the following fields:

1. Increasing coverage of dependents and retired workers.
2. Continued provision of benefits, on a supplemental basis, under employe mutual benefit plans after they have been superseded by arrangements reached through collective bargaining or by employer-sponsored insurance contracts.
3. Greater emphasis upon preventive medicine, evidenced by increased provision for outpatient services.
4. Joint action by a number of smaller unions toward establishment of medical centers or insurance programs, or both.

UNION WELFARE FUNDS GET "ONCE-OVER"

Both the American Federation of Labor and the Congress of Industrial Organizations have pledged broad investigations of suspected misuse of union welfare funds, much in the news of late.

And, the AFL has pledged "strong and wholehearted support" to Government curbs on welfare fund abuses.

According to a recent article in the New York Times, reported in Public Health Economics of November, "A resolution adopted at the convention's final session discarded the Federation's seventy-three-year-old tradition that the Government should keep its hands off internal union problems." Convention delegates representing over 10 million members voted unanimously to support Government curbs on mishandling of union welfare funds.

The newspaper story states: "The convention resolution urged that investigations into welfare rackets eschew politics and give attention to the delinquencies of employers, insurance companies, brokers, agents, state regulatory bodies and physicians receiving fees from welfare plans, as well as the sins of union representatives."

It continues:

"In a number of instances, the AFL said, abuses that have been found to exist stem directly from practices that have long been common in the commercial insurance industry and tolerated by state insurance commissions. These cannot be corrected by investigations or actions directed against organized labor alone. Furthermore, the most serious financial drain upon many health and welfare plans—with no compensating improvement in benefit value or service—has stemmed from systematic overcharges, collusion and other abuses on the part of some elements in the medical profession, motivated by avarice rather than the principle of ethical service at a reasonable fee."

CIO Will Study, Too

Likewise, the CIO has pledged "complete cooperation" with "legitimate" government investigations of alleged welfare fund frauds, and it offered to help write legislation to prevent recurrence of any abuses.

The CIO went a little further, however, and ordered a broad investigation of union welfare funds in an effort to weed out racketeering, according to another article in the *New York Times*. The story stated: "The CIO executive committee established a three-man special committee with power to investigate any allegations of maladministration of welfare or other union funds. The committee will recommend 'prompt remedial action' if abuses are found. The group also will recommend uniform standards for welfare funds and suggest any legislation it believes necessary to protect the funds."

ELECTION BRINGS SPECULATION ON STATUS OF NATIONAL HEALTH INSURANCE

Election of a Democratic-dominated Congress has brought out much guessing on the prospects of possible reintroduction of the national health insurance plan. Because committee chairmanships probably will change hands, this possibility is now more in the forefront. One of the main things the changing of control means, is a fresh look at the health legislation left unpassed, or defeated by the 83rd Congress.

According to a recent issue of the "Washington Report on the Medical Sciences," it is not unlikely that renewed agitation will be felt for passage of a national health insurance bill. The report states: "Reelected to Congress were its foremost champions of national health insurance, Senator James E. Murray (Mont.) and Rep. John D. Dingell (Mich.). . . . Chairmanship of Labor and Public Welfare Committee, which handles most national health bills, is Senator Murray's for the taking. In all probability, however, he will choose to head Interior and Insular Affairs, passing LPW gavel to the next ranking Democrat, who is Senator Lister Hill, of Alabama."

The report states that Senator Hill will then be in a key spot. It notes:

"Alabama's senior Senator not only is due to succeed Senator H. Alexander Smith (R., N. J.) as LPW chairman but his position on Appropriations Committee will be strengthened. In all probability he will serve on, and possibly, head, the sub-committee which acts upon funds for medical research grants, financial support of national hospital expansion under Hill-Burton program, and aid to states for public health. He is an uncompromising opponent of compulsory health insurance, but believes government properly has a role it should not shirk in fostering good medical and hospital care."

FOUR DOCTORS IN HOUSE; TWO DEFEATED

The Congressional elections returned four physicians to the House of Representatives, while two more were defeated for House seats. Those reelected were Drs. Ivor D. Fenton (R., Pa.), Thomas E. Morgan (D., Pa.), A. L. Miller (R., Neb.) and Walter H. Judd (R., Minn.). A fifth, Dr. Will Neal (R., W. Va.), lost out in a bid for a second term to M. G. Burnside, a former member of Congress. And, Rear Admiral Ross T. McIntire, personal physician to the late President Roosevelt and one-time medical director of the American Red Cross, failed in his first bid for a Congressional seat. He was defeated by incumbent Bob Wilson (R., Calif.). Dr. McIntire ran on the Democratic ticket.

MRS. HOBBY PRESSES FOR REINSURANCE AGAIN

Secretary of the Department of Health, Education and Welfare, Mrs. Oveta Culp Hobby, has recently begun to actively beat the drums for the administration's reinsurance plan. Since its defeat, she, as well as President Eisenhower, has asked that it be reconsidered by the 84th Congress, convening in January.

Recently she spoke at the American Life Convention in Chicago, where a large part of her talk was devoted to this subject. Mrs. Hobby said the administration is backing reinsurance because ". . . time is running against those who seek to keep health insurance on a voluntary basis." She added: "We still strongly believe in a bill . . . which seeks to compress the experimentation of the next 20 years into less than half the time through the simple mechanism of a broad sharing of risks. We believe such a bill will . . . nurture rather than weaken the voluntary health insurance concept."

One wonders about the advisability of trying to squeeze "the experimentation of the next 20 years into less than half the time." Wouldn't it be better to go a little more slowly, and allow time for possible mistakes to be avoided by a careful scrutiny of each change before it is made hurriedly?

Besides, there are many excellent reasons for opposing reinsurance, as stated by the American Medical Association:

1. The insurance carriers themselves have all the reinsurance money needed.

2. Voluntary health insurance is making "extremely rapid" progress without reinsurance.

3. Reinsurance would not make uninsurable risks insurable.

4. Without an objectionable subsidy reinsurance would not reduce the cost of insurance or "overcome the inertia of the unwilling buyer."

ACUTE POISONING IN CHILDREN

(Continued from Page 866)

proposed poison control program could be set up initially for the Twin Cities area, rapidly expanded to counties, and ultimately to the entire state of Minnesota.

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Public Health

INFLUENZA AND THE HEALTH DEPARTMENT LABORATORY

The season is now with us when localized outbreaks of influenza may be expected. Observations made in recent times indicate that different types of influenza virus give rise to outbreaks in a fairly regular periodic cycle. There are a variety of influenza virus types and these have been designated as A, B, and C. Within these types there may occur subtypes which differ slightly from types A, B, and C. It is seldom that the same variety of influenza virus is responsible for the outbreak in two successive years. Epidemics of A type influenza tend to recur every two or three years while those of B type recur at four- or five-year intervals. There is little information regarding the occurrence of influenza C. During the influenza outbreak in Minnesota in the winter of 1949-50 the medical laboratories of the Minnesota Department of Health isolated a subtype of type A influenza virus known as A-prime. This A-prime type of virus differed slightly from other known A-prime types of virus. An outbreak of influenza caused by a related A-prime type of virus occurred during the winter of 1952-53. Last winter (1953-54) there was little or no influenza reported. Consequently we need to be on the watch for it this winter.

Because there are variations of the antigenic types of the influenza viruses it is necessary to keep a close check on the type of virus responsible for an outbreak.

The Minnesota Department of Health needs to obtain information as quickly as possible regarding the type of influenza virus that may be causing any specific outbreak. Such information may be useful in making recommendations regarding the use of influenza vaccine and is necessary for epidemiological studies. One method of identifying the type of virus causing the outbreak is by laboratory study of paired blood specimens from persons ill with symptoms compatible with influenza. It is essential that two blood specimens be sent to the laboratory for study, since results of serologic tests are of significance only when a four-fold or greater rise in antibody titer can be demonstrated between specimens taken at different stages of the illness. The first specimen must be taken during the first five days of illness and sent immediately to the Department of Health. A second blood specimen must be taken ten to fourteen days after the first and sent immediately to the Health Department.

Containers for the mailing of blood specimens are available on request from the Minnesota State Department of Health, Division of Medical Laboratories, University Campus, Minneapolis 14, Minnesota. If the laboratory is to supply maximal information, it is essential that the data card accompanying the specimen container be filled in completely and that the directions for collection and submission of specimens be followed. Failure to comply will make it impossible in most cases to perform the tests properly.

Reports of outbreaks of influenza should be made promptly to the State Department of Health with as much clinical and epidemiological information as possible. The dates of onset, number of cases, ages of patients, blood counts, symptoms and any other information the physician can furnish, are all important.

It is of interest to Minnesota physicians who co-operate with the health department in supplying specimens for examination by the laboratory that the data so obtained on influenza outbreaks in this state are reported to the World Health Organization, which gathers epidemiological information from all parts of the world. All over the United States and also in several other countries are laboratories that report outbreaks of influenza and send data on such outbreaks to the WHO. The Minnesota Department of Health operates one of these "collaborating laboratories." Information collected, compared and organized by the WHO is channelled back to the co-operating laboratories. A major purpose of the information thus assembled is to make possible the ultimate preparation and use of vaccines, specifically for the type of disease that is occurring in any geographic area. As the body of knowledge about influenza increases every year, so also does the hope of developing effective vaccines.

Influenza outbreaks in recent years have not been severe, and the high death rate that occurred in the 1918 pandemic have never been repeated. There is, however, no guarantee that this could not happen. It is seldom that the same variety of influenza virus is responsible for an epidemic in two successive years. This tendency of the viruses to be constantly changing increases the difficulty in the control of the disease. That is a major reason for urging physicians to submit specimens to the health department laboratories in all cases of illness believed to be influenza and to report all such cases to the Division of Disease Prevention and Control.

HENRY BAUER, Ph.D.
Director, Division of Medical Laboratories
Minnesota Department of Health

✦ Reports and Announcements ✦

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next scheduled examination (Part I), written examination and review of case histories, for all candidates will be held in various cities of the United States, Canada, and military centers outside the continental United States, on Friday, February 4, 1955. Case Abstracts numbering 20 are to be sent by the candidate to the Secretary as soon as possible after receiving notification of eligibility to the Part I written examination.

For further information, contact the office of the Secretary, Robert L. Faulkner, M.D., 2105 Adelbert Road, Cleveland 6, Ohio.

MINNESOTA SOCIETY OF NEUROLOGY AND PSYCHIATRY

Dr. Reynold A. Jensen, was elected president of the Minnesota Society of Neurology and Psychiatry when the group had its November meeting at the Town and Country Club, Saint Paul. Dr. Royal C. Gray was elected vice president, and Dr. Joseph A. Resch will be secretary-treasurer.

Candidates for active membership who were voted into the Society at the meeting are Dr. A. Margaret Bailey, Dr. G. Wendell Hopkins, Dr. Carl D. Koutsky, Dr. J. Benjamin Lund and Dr. Frances P. Olson.

During the scientific meeting Dr. Gordon R. Kamman spoke on "Critical Evaluation of a Total Push Program for Regressed Schizophrenic Patients in a State Hospital" and Dr. Robert L. Merrick talked on "Greater Superfician Petrosal Neurectomy."

MINNESOTA SOCIETY OF INTERNAL MEDICINE

Officers for the coming year were elected at a Rochester meeting of the Minnesota Society of Internal Medicine. New officers include Dr. H. C. Habein, Rochester, president; Dr. J. F. Borg, Saint Paul, vice president, and, Dr. W. D. Coventry, Duluth, secretary-treasurer.

1955 PAUL-LEWIS AWARD

Dr. Paul D. Boyer, Professor of Chemistry at the University of Minnesota, has been chosen the 1955 recipient of the Paul-Lewis Laboratories Award in Enzyme Chemistry, the Laboratories has announced.

Dr. Boyer's work has been concentrated on problems of mechanisms of reactions in enzymes and on oxidation-reduction of biologically important compounds. The Award is administered by the American Chemical Society and consists of a gold medal and $1,000. The Award will be presented to Dr. Boyer at the 127th national meeting of the American Chemical Society in Cincinnati, Ohio, March 29-April 7, 1955.

UNIVERSITY GETS TB GRANT

The Minnesota Tuberculosis and Health Association has presented a grant of $7,500 in Christmas Seal funds to the University of Minnesota, it has been announced. The grant will finance the H. Longstreet Taylor research project on tuberculosis and related chest diseases under the direction of Dr. J. A. Myers, professor of medicine and public health.

CONTINUATION COURSES

The University of Minnesota announces a continuation course in *Neurology* which will be presented at the Center for Continuation Study, February 7 to 12, 1955. The course, which will be of interest both to physicians in general practice and to those with a special interest in neurology, will stress diagnosis and management of the most frequently-met neurological disorders. Guest speakers will include Dr. G. Milton Shy, National Institute of Neurological Diseases and Blindness; Dr. Howard Fabing, vice president, American Academy of Neurology, Cincinnati; and Dr. Edward B. Schlesinger, clinical assistant professor of neurosurgery, Columbia University College of Physicians and Surgeons, New York City. The annual Johnston lecture will be presented by Dr. Seymour S. Kety, also of the National Institute of Neurological Diseases and Blindness. The course will be presented under the joint direction of Dr. A. B. Baker, professor and head of Division of Neurology, and Dr. William T. Peyton, director, Division of Neurosurgery, University of Minnesota Medical School.

The University of Minnesota and the Minnesota Division of the American Cancer Society will join in the presentation of a continuation course in *Cancer Detection for General Physicians* next February 17 to 19, 1955, at the Center for Continuation Study. This year's program will consist of a series of lectures and informal discussions concerning cancer detection techniques. Those registrants wishing to observe and participate in certain examination procedures will be given an opportunity to do so.

RENEWAL OF NURSES' LICENSES

Minnesota registered nurses who are currently renewed for 1954 and plan to be employed as registered nurses in this state in 1955, are reminded that renewal of license is due by December 31, 1954, the Minnesota State Board of Examiners has announced. Those who are currently renewed and expect to retire from employment as registered nurses in Minnesota (by marriage, moving to another state, or other reasons) and do not plan to be employed in Minnesota at any time

(Continued on Page 902)

Woman's Auxiliary

PRESIDENT'S LETTER

Greeting old friends and meeting new friends on the local level has been a pleasurable and stimulating experience this fall. Regional Advisors Mrs. O. M. Heiberg, Mrs. T. R. Fritsche, and Mrs. W. E. Wellman arranged regional meetings in Fairmont, New Ulm and Rochester. The programs for the meetings were similar in effect, designed to bring the members up to date on problems and responsibilities of the auxiliary. After the meeting in Fairmont, we were invited to attend the Martin County Hospital Auxiliary meeting at which our own Maude Halpern* gave a superb presentation on "Working Together for Health and Safety." Earl Dresser, Assistant Administrator of Asbury Hospital in Minneapolis, was guest speaker.

Your president presided at all the meetings, bringing up-to-date auxiliary news to the members after welcoming the women in behalf of the state auxiliary. Legislation news and mental health news was presented. We are urged to offer our services to the schools in our community so as to help cope with the added responsibilities that have resulted from the overcrowded schools. It is the general opinion that problems of the school and the home contribute greatly to the increase of the mentally ill among the school children.

Mrs. H. H. Fesler, president-elect, addressed the women on organization news, and Mrs. C. L. Sheedy of Austin addressed the group on Today's Health and the American Medical Education Foundation. We are urged to participate, also, in "Operation Christmas" which calls for one or more subscriptions to *Today's Health* as a gift from a doctor's wife to a layman. Teachers, new mothers, beauty shops, barber shops, schools, are excellent for placing our health magazine.

We are urged to support the American Medical Education Foundation each year. As our State University has benefited from this fund far beyond the total contributions from our state to the amount of $20,000.00 as reported last year, we should make this a yearly obligation.

In Rochester, Mrs. Stanley Peterson of Austin addressed the women on "Allied Medical Careers," and Mrs. Harold Wahlquist addressed the group on "Health Days."

On November 5, your president participated on the Question-and-Answer Panel of the all-day press conference sponsored by the *Minneapolis Star and Tribune* at the Radisson Hotel. This was a most interesting day. Many tips on club news were given.

An excellent address on "Forces at Work in the Middle East" was given by Yahya Armajani, Professor of History, Macalester College, Saint Paul. "1954 Post Mortems" was presented by Doris Fleeson, Washington correspondent.

*Mrs. Halpern's address will be published in the January issue.

Chairmen should be called "Press" and not Publicity Chairmen. Don't sit on news. And that reminds me that our own MINNESOTA MEDICINE welcomes auxiliary news of all activities of the county auxiliaries. Mail this news to Mrs. L. Raymond Scherer, 1930 Irving Place, Minneapolis 5, Minnesota.

Congratulations to the newly re-organized Kandiyohi-Swift-Meeker County Auxiliary. We wish the members a most successful year.

With the holiday season approaching, I wish all the members of the auxiliary and every doctor's wife, a Merry Christmas and a Happy New Year.

MRS. PETER S. RUDIE, *President*
Woman's Auxiliary to the Minnesota
State Medical Association

RAMSEY ACTIVITIES

Mrs. L. T. Simons

Mrs. W. P. Gardner called a board meeting on November 15 in the Ramsey County Medical Society library. Committee reports were given and plans for the coming meetings were discussed. Cancer dressings were made during and following the meeting.

Mrs. W. A. Gleason, press chairman, and Mrs. W. P. Gardner, president, attended the *Minneapolis Star and Tribune* press conference in the Radisson Hotel, November 5. Mrs. P. S. Rudie, state president, was a member of the panel. Doris Fleeson, Washington correspondent, presented an interesting discussion on trends in the news.

NEW COUNTY AUXILIARY ORGANIZED

Mrs. Harold H. Fesler,
State Organization Chairman

Thursday, October 21, was an important date for the Woman's Auxiliary to the Minnesota State Medical Association. A new county society was organized, at Willmar. Mrs. P. S. Rudie, state president, attended a meeting in September, in Willmar, and discussed the possibility of forming an auxiliary.

We were invited to attend a meeting at the Country Club with the doctors, which was most enjoyable. While they held their meeting, following the dinner, the women retired to the home of Mrs. L. C. Gilman,

Mrs. Rudie talked on the history, activities and program of the auxiliary and I, as organization chairman, gave the aim and purpose. A motion was made and thoroughly discussed, that a charter be drawn up, and it was unanimously decided to form the Kandiyohi-Swift-Meeker County Auxiliary—Mrs. Charles Waas served as temporary secretary. Officers were elected with Mrs. Willard Peterson as president. Fifteen attractive, young and enthusiastic members signed the

(Continued on Page 903)

In Memoriam

CHARLES M. ADKINS

Dr. Charles M. Adkins, eighty-two years old, died at his home in Thief River Falls after a long illness November 2, 1954. He became a life member of the Minnesota State Medical Association after forty-one years of membership in the Red River Valley Medical Society in 1951.

Dr. Adkins served as Pennington County coroner for many years and spent three years in the Philippines during the Spanish American War.

He was born in Wayne County, West Virginia, and went to high school at Fairhaven, Mississippi. He was graduated in medicine from the Kansas City Medical College of the University of Kansas and served his internship at the Kansas City General Hospital. He practiced at Ogema from 1909 to 1912 and at Grygla for short periods. Most of his professional career was spent at Thief River Falls where he was city physician and local surgeon for the Great Northern Railway as well as director of Physicians' Hospital.

Survivors are his wife, the former Alice Solem; one daughter, Arda, Thief River Falls; five sons, two of whom are physicians, Dr. Galen Adkins of Anoka, and Dr. Douglas Adkins of Minneapolis, Jack Adkins, also of Minneapolis, and Peter and Caylor Adkins of Thief River Falls; one sister, Mrs. Dellis Wisehard of Cass Lake; and one brother, Jones Adkins of Minneapolis.

WILLARD E. ARCHER

Dr. Willard E. Archer died of a heart attack at the age of fifty-five, October 13, 1954, at Falls Creek, Wisconsin. He was on leave from the staff of the Anoka State Hospital.

Dr. Archer was born at West Union, Iowa, and was graduated in medicine from Marquette University. He served his internship at St. Elizabeth's Hospital in Appleton, Wisconsin, and spent two periods in post-graduate work at the Cook County Post-Graduate School in Chicago. He practiced for twelve years at Dale, Wisconsin, and for fourteen years specializing in obstetrics and gynecology at Appleton. After that he spent three years in neurology and psychiatry at the Veterans Administration Hospitals at Tomah, Wisconsin, and Hines, Illinois, and joined the staff of the Anoka Hospital in 1952.

Dr. Archer was a member of the Hennepin County Medical Society, the Minnesota State Medical Association and the American Medical Association, and he had served as president of the Outagamie County Medical Society of Wisconsin.

Survivors include his wife, the former Florence Heiserman; two daughters, Mrs. M. D. Davis, Bremerton, Washington, and Patricia, a nurse at Methodist Hospital, Madison, Wisconsin; and one son, Corporal Thomas Archer, Fort Ord, California.

HERMANUS DE BOER

Dr. Hermanus De Boer of Edgerton died at Pipestone Hospital October 27, 1954. He was eighty-three years old and had practiced at Edgerton since 1906.

Until he retired from active practice in 1946, Dr. De Boer had played an active part in the life of the community. In the early days he made hundreds of night calls in the countryside by team and bob-sled and he had brought close to three thousand infants into the world, many of whom were present at a special "Dr. De Boer Day," celebrated under sponsorship of the Edgerton Civic Club in March, 1949. For 42 years he served as a member of the Edgerton Board of Health and for 33 years on the Board of Education. He was mayor of the village for 22 years.

He was also a life member of the Minnesota State Medical Association, a member of the American Medical Association, and one-time president of the Southwestern Minnesota Medical Society. He was a staff member of the Ashton Memorial Hospital at Pipestone and of the Home Hospital at Slayton.

Dr. De Boer began a varied and adventurous career as a lawyer, having received his law degree at Kent College of Law in Chicago in 1894. He practiced law in Chicago for two years and then left for South Africa to open a steam laundry there. He was joined by the first Mrs. De Boer, the former Dora Sietsema, but they returned to Chicago shortly afterwards and Dr. De Boer entered the National Medical University of Chicago from which he was graduated in 1903. He practiced for three years in Chicago before going to Edgerton.

The first Mrs. De Boer died in 1949 and Dr. De Boer remarried in 1953. He is survived by his widow and an adopted son, Theo, of Plainview.

JOSEPH H. DUDLEY

Dr. Joseph H. Dudley, physician and leader for many years in civic affairs in Windom, died October 21, 1954, at the age of eighty-two. He had been a patient at the Windom hospital for more than a year and had been in ill health for several years.

Dr. Dudley opened his practice in Windom in 1909 after practicing for ten years following graduation from Rush Medical College in Chicago, at Heron Lake.

He headed the Windom Civic and Commerce Association as president for many years and served as judge of probate for Cottonwood county from 1940 to 1952. A stalwart Republican in politics, he also served as chairman of the county congressional committee at one time. He left his practice three times, in 1906, 1916 and 1930, to take post-graduate training in Chicago, and was a member of the Southwestern Minnesota Medical Society, the Minnesota State Medical Association and the American Medical Association. He was also a member of the Windom Presbyterian Church, a Mason and a

member of the Woodmen of America, the Royal Arcanum, the Modern Brotherhood of America and the Royal Neighbors.

The only survivors are an adopted son, P. T. Dudley of St. Louis, Missouri; two grandchildren; and a nephew, Art Dudley of St. James.

EDWARD THOMAS FITZGERALD

Dr. Edward T. Fitzgerald, who practiced for forty years at Morris, died October 13, 1954, at the age of seventy-nine. He was a life member of the Minnesota State Medical Association and had practiced at Morris from 1911 until his retirement in 1940 because of ill health.

Dr. Fitzgerald was born at Boone, Iowa, and was educated in Boone County schools, the Iowa City Academy and Creighton Medical College at Omaha from which he was graduated in 1907. He practiced at LeMars, Iowa, for three years before going to Morris.

He served on the State Board of Health from 1936 to 1940, and was a member of the West Central Minnesota Medical Society, the Minnesota State Medical Association, and the American Medical Association.

GORDON BALGARNIE NEW

Dr. Gordon B. New, distinguished laryngologist and plastic surgeon and one of the pioneers in the organization of the Mayo Clinic, died at Methodist Hospital in Rochester after a short illness, October 27, 1954.

Dr. New was sixty-eight years old at the time of his death. He retired from the clinic in 1950 and had been extensively honored in this country and abroad for his contributions to the field of plastic surgery. One of the pioneers in his field, he was also an international authority on tumors of the nose and throat and held top offices in many medical and surgical organizations. In 1952, he was awarded the top honor that can be given to a surgeon by the American Society of Plastic and Reconstructive Surgery. He received many awards for exhibits on correction of facial deformities and on cancer of the mouth.

Dr. New was born in Hamilton, Ontario, Canada, in 1885. He received a degree in dental surgery from the University of Toronto in 1906, the degree of doctor of medicine in 1915. He served an internship in the City Hospital of Hamilton and came to Rochester in 1919 as head of the section of postoperative treatment and as assistant in laryngology and rhinology, oral and plastic surgery.

When the Mayo Foundation was established as part of the graduate school of the University of Minnesota in 1915, Dr. New was appointed assistant professor of rhinology, laryngology and stomatology. Later he became professor of laryngology.

He was elected president of the American Association of Oral and Plastic Surgeons in 1931, and president of the American Academy of Ophthalmology and Otolaryngology in 1946. In 1947, he was vice president of the American College of Surgeons. He was a member of a long list of scientific societies as well as the Olmsted-Houston-Fillmore-Dodge County Medical Society, the Minnesota State Medical Association and the American Medical Association.

He is survived by his wife, the former Ethel Bailey; two sons, Gordon of Solvang, California, and Robert of Lakewood, Ohio; and two grandsons. A daughter, Marian Elizabeth, and two brothers preceded him in death.

JAMES ALFRED SANFORD

Dr. James A. Sanford, who practiced at Farmington and Savage for more than forty years, died at his winter home in Hot Springs, Arkansas, November 15, 1954. He was seventy-five years old, a life member of the Minnesota State Medical Association and a member, since 1951, of its Fifty Club.

Dr. Sanford was a leading figure in the community for many years. He built the modern hospital at Farmington, helped found its telephone company and served on village councils, which gave the town sewer and water, electricity and paved streets. He was regarded with great pride by fellow townsmen as the typical "country doctor" and he had recently written a book about his experiences entitled "The General Practitioner" which is to be published soon by Doubleday & Co., of Garden City, New York.

Dr. Sanford was born at Alderley, Wisconsin, in 1879 and went to high school in Hartford, Wisconsin. He took his medical training at the University of Minnesota, from which he was graduated in 1901. He practiced in New Market for five years and in Minneapolis for three years before moving to Farmington. He was a member of the Scott-Carver Medical Society and the American Medical Association, as well as the Minnesota State Medical Association.

Surviving are his wife, Lillian; a daughter, Mrs. Helen Rondestvedt of Sauk Centre; one grandson, four granddaughters and his daughter-in-law, Mrs. Lucy Sanford, widow of his son, James, of Farmington.

RENEWAL OF NURSES' LICENSES

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in 1955 should request placement in the non-practicing status, to begin in 1955.

Minnesota registered nurses who now are in the non-practicing status but who return to employment must renew their license prior to employment. Applications for 1955 renewal or non-practicing status are attached to the 1954 renewal certificate. Applications and one dollar fee (only for those renewing for 1955) may be sent to the Minnesota State Board of Examiners of Nurses, 700 Minnesota Building, St. Paul 1, Minnesota. Money order or certified bank check is the preferred means of payment. *All* Minnesota Licensed Practical Nurses *must* renew their licenses on a calendar year basis.

Letters to the Editor

Arthur H. Wells, M.D.
Minnesota Medicine
519 East First St.
Duluh 5, Minnesota

Dear Doctor Wells:

Jaundice Associated with the Administration of Chlorpromazine (Thorazine). The correct etiologic diagnosis is, of course, essential for the proper treatment of the jaundiced patient. Among other important factors the critical decision of surgical intervention depends upon the accuracy of the differential diagnosis. Unfortunately the problem has often been difficult and the recent reports of jaundice associated with the administration of Chlorpromazine (Thorazine) would seem to further complicate the diagnostic difficulties.

Lemire and Mitchell (Proceedings of the Central Society for Clinical Research, 27:70, 1954) have recently reported three cases of "regurgitation type jaundice" associated with Chlorpromazine (Thorazine) administration. In each case evaluation of liver function revealed a hyperbilirubinemia, an increase in the alkaline phosphatase activity and a normal cephaline cholesterol flocculation. Since the drug was not considered etiologically involved and since the evaluation of liver function alteration suggested extrabiliary tract obstruction all three patients underwent laparotomy. There was no evidence of such extrahepatic obstruction. Liver biopsies revealed plugging of the bile canaliculi with bile. The parenchymal cells showed brownish pigmentation deposition but were otherwise normal. There were some areas of spotty infiltration of inflammatory cells within the liver lobule.

There are, of course, many drugs which are hepatotoxic; however, for the most part these agents, if they result in the development of jaundice, are associated with liver function abnormalities normally considered compatible with hepatocellular injury. Chlorpromazine (Thorazine) differs markedly in this respect in that it is capable of producing jaundice with liver function abnormalities ordinarily considered compatible with extrabiliary tract obstruction or so-called surgical jaundice. The following brief case report illustrates another instance of jaundice associated with Chlorpromazine (Thorazine) administration.

Case Report.—A 67-year-old white woman was known to have hypertension, coronary artery disease and moderately severe chronic anxiety. It was apparent that much of her disability was related to her neuropsychiatric problems. She was inadequately controlled by mild barbiturate sedatives and on October 20, 1954, she was started on Chlorpromazine (Thorazine) in a dose of 25 mgms. three times daily. There was considerable subjective improvement; however, on November 10, 1954, twenty-two days after the institution of drug therapy, she was noted to have definite icterus of the sclera. There was no associated pain and initially there was no anorexia, nausea or vomiting. There was rather marked pruritus of the skin. The liver edge was palpable approximately 3 cm. below the right costal margin but was not tender. The spleen was not enlarged. Liver function studies revealed the serum bilirubin to be 5.1 mgms. per cent in one minute with a total of 7.8 mgms. per cent. The cephalin cholesterol flocculation test was negative at 24 and 48 hours. The alkaline phosphatase activity of the serum was 52 King-Armstrong units.

In the above recorded case the findings were considered identical to those noted by Lemire and Mitchell and therapy was to consist of simple withdrawal of the Chlorpromazine (Thorazine) administration. To date it would appear that this syndrome has been primarily noted in elderly patients and present information suggests that the process is reversible. The importance of the syndrome is not so much in reporting another drug capable of producing jaundice but in recognizing that the liver function abnormalities are not those of hepatocellular damage but rather those commonly associated with extrahepatic biliary tract obstruction. It is most important that these patients be spared unnecessary surgical intervention.

Very truly yours,
WYMAN E. JACOBSON, M.D.
St. Louis Park Medical Center
Department of Medicine

November 15, 1954
St. Louis Park

RAUWOLFIA

(Continued from Page 872)

psychotherapy against anxiety, *causeless* anxiety —the master symptom of all of us, belonging as we all do, to that part of the population that may be called the non-institutionalized insane.

Rauwolfia poses a question: Are many of these anxieties prodromal symptoms of deeper mental illness, of psychoses?

If they are, then with Rauwolfia (and chlorpromazine too) general practitioners are likely to wake up to find themselves in the front line of the fight, not to build more mental hospitals, but to begin to empty present institutions for the insane.

This is the glory of American medicine: that there is no government or totalitarian authority telling physicians they've got to save lives of hypertensives and keep the patients from going crazy—or else. Now that Rauwolfia is showing that these things can often be done, it's purely between the doctor and his God or if he hasn't a God, his conscience; it's the individual doctor's lonely responsibility.

NEW COUNTY AUXILIARY ORGANIZED

(Continued from Page 900)

charter and we are most happy to welcome them, one more active group to strengthen our organization.

Mrs. H. E. Bakkila of Duluth and Mrs. Charles Wass of Saint Paul, both past state presidents of the auxiliary, Mrs. John Dordal of Sacred Heart, first vice president, Mrs. E. R. Hudec of Echo, regional advisor, and Mrs. M. I. Hauge of Clarkfield, chairman of Today's Health, were all present at this meeting and enjoyed the thrill as much as I. We are happy to welcome our new friends and wish them the best of luck, success and happiness.

Of General Interest

Several Mayo Clinic staff members attended national meetings last month. **Dr. N. W. Barker** and **Dr. E. A. Hines, Jr.,** were present at the meeting of the American Society for the Study of Arteriosclerosis in Chicago. Dr. Barker is a member of the society's board of directors. **Dr. H. B. Burchell** attended a meeting of the research committee of the American Heart Association in Boston and **Dr. R. Q. Royer** was in Hollywood, Florida, for the meeting of the American Society of Oral Surgeons. Present at a session of the American Board of Preventive Medicine in New York City was **Dr. J. H. Tillisch.**

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Dr. R. O. Sather of Crookston was in Chicago for a course at the University of Chicago on recent advances in the field of internal medicine.

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Dr. Roberts Zemmers of Duluth was one of twenty foreign-born persons who met President Eisenhower in Washington, D. C., last month. The group received naturalization papers as citizens that day. Dr. Zemmers, a native Latvian, is associate anesthesiologist at St. Luke's Hospital.

* * *

New president-elect of the American Society of Plastic and Reconstructive Surgery is **Dr. Frederick A. Figi** of the Mayo Clinic, Rochester. Dr. Figi will be installed into the office, now held by **Dr. William G. Hamm** of Atlanta, at next year's meeting.

* * *

Dr. R. P. Hallin of Worthington has been elected president of the Southwestern Minnesota Medical Society. Also elected at the annual meeting at Pipestone were **Dr. H. A. Christiansen,** Jackson, president-elect; **Dr. Victor Doman,** Lakefield, vice president; and **Dr. O. M. Heiberg,** Worthington, secretary. Named delegates to the state medical convention were **Dr. John Lohmann,** Pipestone, and Dr. Heiberg. Alternates are Dr. Hallin and Dr. Christiansen. Voted into the society by transfer were **Dr. Milton Plucker** and **Dr. Christian A. Rohrer,** both of Worthington.

* * *

Four Mayo Clinic physicians participated in national meetings the early part of November. **Dr. R. E. Fricke** gave a paper on the subject "Late Recurrences of Pelvic Cancer: With Description of an Unusual Case" at a meeting of the American Therapeutic Society in St. Louis. Speaking before the Interim Scientific session of the American Rheumatism Association in Bethesda, Maryland, was **Dr. L. E. Ward,** who spoke on "Effects of Aldosterone and 9-Alpha-flourohydrocortisone Acetate Administered to Rheumatoid Patients." **Dr. K. N. Ogle** was in Toronto to attend a joint meeting of the Armed Forces-National Research Council Vision Committee and the Vision Panel of the Canadian Defense Research Board. **Dr. H. F. Polley** attended a meeting of the American Rheumatism Association in Bethesda, Maryland.

* * *

Dr. W. O. Lundberg, director of the University of Minnesota's Hormel Institute, has been invited to be an original signer of a proclamation establishing an international society for fat science. Only two other Americans, **Dr. A. R. Baldwin,** director of research, Cargill, Inc., and **C. E. Morris** of Armour and Co., president of the American Oil Chemists Society, have been asked to serve as sponsors of the organization. Scientists from sixteen countries have agreed to serve as sponsors for the organization, which will include men working in the fields of chemistry, biology, medicine, engineering and agriculture.

* * *

Dr. John Fischer of Owatonna has been elected coronor of Steele County. He was endorsed by the Steele County Medical Society.

* * *

Dr. A. M. Struthers, a fellow in plastic surgery in the Mayo Foundation, has been awarded second prize in the annual essay contest of the Foundation of the American Society of Plastic and Reconstructive Surgery. Dr. Struthers won the prize, a three-month scholarship for study in plastic surgery centers here and abroad, for a paper on the topic "An Experimental Study of Polyvinyl Sponge as a Substitute for Bone."

* * *

"The RH Factor in Practice" was the topic of a talk given by **Dr. James P. O'Keefe** of St. Cloud at a meeting of the Kandiyohi-Swift-Meeker Tri-County Medical Society. The dinner session was held in Willmar.

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Dr. Albert J. Schroeder of Minneapolis addressed the Specialty Section of the Wisconsin State Medical Society in Milwaukee on the topic "Infectious Croup." Dr. Schroeder also conducted a round-table discussion on the subject "Hemolytic Disease of the Newborn."

* * *

Newly-elected chief of staff of the Waconia Community Hospital is **Dr. Leighton W. Larson,** Waconia. Vice chief of staff will be **Dr. Joseph Selmo** of Norwood. **Dr. J. W. Clarke,** Watertown, was elected secretary-treasurer. Executive committee members are **Dr. R. E. Pogue** of Watertown and **Dr. R. S. Johnson** of Excelsior.

* * *

Dr. D. H. Petersen of St. Olaf College, Northfield, and **Dr. J. W. Hanson,** Carleton College, Northfield, attended the regional meeting of the

American College Health Association recently at Luther College in Decorah, Iowa.

* * *

At home in Minneapolis are **Dr. and Mrs. Karel Bedrich Absolon,** who were recently married in St. Ignatius Church. The bride, the former Mary Bendix, is the daughter of **Dr. and Mrs. Lester H. Bendix** of Annandale. Dr. Absolon's parents are **Dr. and Mrs. Karel Absolon** of Brno, Czechoslovakia.

* * *

Mayo Clinic staff members who attended the International Symposium on hypophyseal growth hormone: nature and actions, in Detroit, were **Dr. A. Albert, Dr. E. V. Flock** and **Dr. A. B. Hayles.** The symposium was sponsored by Henry Ford Hospital.

* * *

"Social Work and Medicine" was the topic of the regional Minnesota Welfare conference last month in St. Paul. Speakers for the meeting included **Dr. Dale C. Cameron,** State Department of Public Welfare medical director; **Dr. William Maloney,** assistant dean of the University of Minnesota Medical School; **Dr. Lyle Hay,** surgeon and chief of staff at Mount Sinai Hospital, Minneapolis; and **Dr. Robert Mooney,** St. Paul internist.

* * *

University of Minnesota board of regents has promoted **Dr. Robert A. Good** to American Legion memorial research professor, replacing **Dr. Lewis Thomas,** who resigned to go to New York University. Dr. Good, a graduate of the University, was associate professor of pediatrics. He has been a fellow of the National Foundation for Infantile Paralysis, a visiting investigator at the Rockefeller Institute of Medical Research and holder of a Helen Hay Whitney Foundation fellowship. At the University he has been Markle Foundation scholar in medical sciences.

* * *

Dr. and Mrs. Merrill D. Chesler of Minneapolis attended the meeting of the American Society of Plastic and Reconstructive Surgery at Hollywood, California, in October. Dr. Chesler was elected to membership in the Society at the meeting.

* * *

Elected to membership of the International Society of Hematology was **Dr. Frances E. Schaar** of Minneapolis. The selection was made at the Fifth Congress of the Society in Paris, France.

* * *

Dr. Russell M. Wilder, former professor and head of the Mayo Clinic's Department of Medicine, has been awarded the American Medical Association's Joseph Goldberger award in clinic nutrition. The award was received by Dr. Wilder at ceremonies in Washington, where the annual meeting of the Food and Nutrition Board of the National Research Council took place.

* * *

One of Bemidji's pioneer physicians, **Dr. D. F. McCann,** has retired from the practice he founded in 1916. Dr. and Mrs. McCann have left for California. Dr. McCann, sixty-five, was graduated from Columbia University College of Physicians and Surgeons and served his internship in New York hospitals. The Bemidji practice was interrupted when Dr. McCann served in World War I as a regimental surgeon, and for several years when the McCanns resided in McCloud, California. In Bemidji, Dr. McCann founded the Bemidji Clinic with **Dr. T. P. Groschupf** and **Dr. D. D. Whittemore.**

* * *

Dr. and Mrs. Harold D. Nagel have left Waconia to reside in Minneapolis. The Nagels had resided in Waconia for thirty-one years. They turned over their hospital, built in 1940, and nurses' home to the Waconia Community Hospital Association.

* * *

Life of a Rice County pioneer doctor, **Dr. N. M. Bemis,** was reviewed at a recent meeting of the Rice County Historical Society at Faribault. The paper was prepared by **Dr. Adolph Hanson** of Faribault. Dr. Bemis, the first doctor to reside in Rice County, practiced there from 1856 to 1891, date of his death.

* * *

Five Minnesota physicians have been re-elected to the board of directors of the Minnesota Tuberculosis and Health Association. They are **Dr. Leon H. Flancher** of Crookston, **Dr. Charles Sheppard** of Hutchinson, **Dr. V. M. Johnson** of Dawson, **Dr. W. H. Valentine** of Tracy, and **Dr. A. L. Vadheim** of Tyler. The men are officers in their county Christmas Seal organizations. Another Crookstonite elected to the Association was Mrs. M. O. Oppegaard. The board members were re-elected at the Seal organization's annual session in St. Paul.

* * *

Dr. N. W. Barker was elected president of the voting staff of the Mayo Clinic at the annual meeting of the group in November. He succeeds **Dr. F. J. Heck.** Other officers named were **Dr. L. E. Prickman,** vice president; **Dr. D. G. Pugh,** re-elected secretary;**Dr. L. D. Vaughn,** first councilor, and **Dr. R. R. Kierland,** second councilor. In addition to the election, the staff members presented emeritus awards to **Drs. S. W. Harrington, A. B. Nevling, F. P. Moersch** and **H. W. Woltmann,** who have retired during the year. Action was also taken confirming positions of **Dr. H. K. Gray** and **Mr. J. W. Hartwick** on the board of governors. Introduced as new members of the voting staff were **Drs. R. O. Brandenburg, A. J. Bruwer, R. L. Faucett, R. W. Gifford, Jr., N. O. Hanson, H. F. Helmholz, Jr., F. T. Maher, W. F. McFuckin, R. H. Miller, A. L. Orvis, J. E. Osborn, R. J. Reitemeier, R. A. Rovelstad, R. E. Symmonds** and **P. E. Zollman.**

* * *

Dr. L. L. Sogge of Windom, Cottonwood County Christmas Seal chairman for over thirty years, was

honored at a district workshop meeting of Seal committees at Worthington. A medal, the first to be awarded in Minnesota, was given Dr. Sogge, on behalf of the National Tuberculosis Association, by **Dr. S. A. Slater,** principal speaker at the meeting. Dr. Sogge was cited for his fifty years of service in tuberculosis work.

* * *

"Fluoridation of Water" was the topic of a talk given by **Dr. Dean Soderberg,** Duluth, at a combined meeting of the Second District Minnesota Nurses and Practical Nurses groups. The meeting was held in St. Mary's Hospital.

* * *

Dr. Robert Kallsen, who has been in Crosby since June in association with **Dr. C. M. Marshall** and **Dr. R. E. Kelley,** will leave for California to take up practice. Returning to Crosby is **Dr. James Nixon,** who has been on duty with the Navy in California and overseas.

* * *

Dr. Fred W. Wittich, Minneapolis, president of the International Association of Allergology, spoke at the first graduate symposium on geriatric medicine last month in New York. Dr. Wittich also participated in a panel discussion on allergies in older patients.

* * *

Representing western Minnesota at the American College of Surgeons sessions in Atlantic City, New Jersey, was **Dr. I. L. Oliver** of Graceville. Dr. Oliver also visited his son, Robert, at Trinity College, Hartford, Connecticut.

* * *

Dr. Kenneth R. Larson of St. Paul attended sessions of the third Pan-American Congress on Endocrinology, November 21-27, in Santiago, Chile. Dr. Larson and Mrs. Larson also visited other Central and South American points during their trip.

* * *

Guest lecturer at the National University, Bogota, Columbia, South America, for a three-week period was **Dr. Harold O. Peterson** of St. Paul. Dr. Peterson, who was accompanied by his wife, lectured on the technique of myelography from November 19 to December 8. Dr. Peterson is clinical professor of radiology at the University of Minnesota and director of radiology at the Charles T. Miller and Children's Hospitals in St. Paul. The Petersons visited in Rio de Janiero, Brazil, and Lima, Peru, en route to Columbia.

* * *

Dr. John Falls of Chicago, Illinois, has joined the staff of the Interstate Clinic at Red Wing. Dr. Falls, a graduate of the University of Illinois Medical School, will specialize in obstetrics and gynecology in association with **Dr. Grant Hartnagel.**

* * *

Dr. Charlotte J. Morrison has reopened offices at 911 Medical Arts Building, Minneapolis. Her practice is limited to pediatrics.

Dr. Wesley W. Spink, professor of medicine at the University of Minnesota, was elected recently to a two-year term as president of the Minnesota Medical Foundation, succeeding **Dr. Owen H. Wangensteen,** head of surgery at the University. Vice President is **Dr. R. S. Ylvisaker,** clinical associate professor of medicine. **Dr. Robert B. Howard,** director of postgraduate medical education, succeeded Dr. Spink as secretary-treasurer.

* * *

Patients of Sunnyrest Sanatorium at Crookston honored **Dr. L. H. Flancher,** superintendent, at a farewell party recently. Dr. Flancher will assume the post of public health superintendent for the district. Sunnyrest Sanatorium has been merged with Oak Park Sanatorium at Thief River Falls. **Dr. Baldwin Borreson,** superintendent of Oak Park for almost twenty years, will take charge of the combined groups at Crookston. The combined district, which will be headed by Dr. Flancher, includes Polk, Norman, Pennington, Red Lake, Marshall and Roseau counties.

* * *

Dr. Ralph Rossen, head of Hastings State Hospital, has resigned to take over a position as director of a private mental research project. Dr. Rossen, Minnesota's first mental health commissioner, will become director of the L. E. Phillips psycho-biological research fund project at Mount Sinai Hospital, Minneapolis. Dr. Rossen took over his new duties the first of December. **Dr. Jorge Lazarte** is acting head of Hastings Hospital.

* * *.

Guest speaker at a clinical conference in November in Glencoe was **Dr. William F. Maloney,** assistant dean of the University of Minnesota Medical School. Dr. Maloney spoke on heart disease.

* * *

Dr. Donald McMullen, chief of the department of medical zoology, Walter Reed Army medical center, Washington, D. C., was principal speaker at a University of Minnesota continuation course on clinical microscopy and parasitology. The course was held in November.

* * *

Training with the Air Force at Montgomery, Alabama, is **Dr. Rolf Daehlin,** formerly of Fergus Falls. Dr. Daehlin will be stationed at Spokane, Washington, after completing training. He will be in charge of pediatrics at Fairchild Air Force Base.

* * *

Guest speaker at a meeting of the Duluth District Dental Society and the St. Louis County Medical Society in November was **Dr. Thomas J. Dry** of Rochester. Dr. Dry spoke on "Progress in the Treatment of Heart Disease by Surgical Means." **Dr. Alex Kellem,** president of the dental group, presided at the meeting.

* * *

Dr. R. P. Pearsall, Virginia physician and city health officer, was commended by the Virginia Red Cross branch board of directors for his outstanding

service to the Red Cross. Recognition was made at a November board meeting.

* * *

Two Blue Earth physicians, **Dr. D. V. Smith** and **Dr. George Drexler,** participated in postgraduate courses in November in Chicago.

NEW LOCATIONS

Dr. Ivan Sawaryniuk, who has been associated with Dr. H. D. Nagel in Waconia, will set up practice there in the Diessner Building. Dr. Sawaryniuk, a graduate of the University of Frankfurt, Germany, came to Waconia from Mt. Sinai Hospital in Minneapolis.

* * *

Dr. V. A. Schulberg, formerly of Arlington, will be associated with **Dr. D. O. Olson** at Gaylord. Dr. Schulberg has been in Los Angeles, California, for the past two years. He is a graduate of the University of Minnesota.

* * *

Dr. Carl Heinzerling of Minneapolis will be associated with **Dr. R. E. Pogue** and **Dr. J. W. Clarke** in their Watertown medical practice. Dr. Heinzerling, a University of Minnesota Medical School graduate, interned at Mt. Sinai Hospital.

* * *

Dr. Dwain Paal of Currie has become associated with Dr. Leonard Lang in the Medical Arts Building, Minneapolis.

* * *

Now associated with **Dr. E. R. Hudec** at Echo is **Dr. David Sontag,** who has been with the Veterans Administration Hospital at Los Angeles, California.

CAREER: MEDICAL TECHNOLOGY

(Continued from Page 888)

Physicians are strongly urged to make free use of these films. The two societies sponsoring their use will be glad to co-operate in furnishing a pathologist or a medical technologist or both at the showing.

As already indicated, the medical technologist occupies an important place on the medical team. Anything that can be done by the physicians of Minnesota to help eradicate the present shortage of qualified medical technologists will improve the practice of medicine and will redound to the ultimate benefit of the patient.

MINNESOTA BLUE CROSS-BLUE SHIELD

Recent reports regarding Blue Shield utilization during the first eight months of 1954 show a distinct increase over the same period of 1952 and 1953 in the number of services for which Blue Shield has provided benefits. An analysis of the utilization report for this period, compared with the reports for the first eight months of 1952 and 1953, shows not only this marked increase in the number of services covered by Blue Shield, but also a definite increase in the average payment for such services.

The most significant increase in the average cost per service is found in medical care, where services rose from 17,780 in the first eight months of 1952 to 23,432 for the same period in 1954, or a 32 per cent increase. Here the average Blue Shield payment for medical service increased from $22.93 in the first eight months of 1952, to $29.33 for the first eight months of 1954, an increase of 28 per cent.

Another distinct increase is in obstetrical care. Obstetrical services paid by Blue Shield rose from 8,732 in the first eight months of 1952 to 9,750 in the same period in 1954. Greater benefits which became effective on January 1, 1954, combined with the reduction of the waiting period, increased both the number of services and the payments made to doctors by approximately 14 per cent.

Some increase in the number of services can be attributed to increased enrollment, but actually additional enrollment is proportionately less than the increase in the number of services.

Effective October 1, 1954, more than 50,000 Minnesota Blue Cross $25 Deductible non-group subscribers were converted to the new $25 Deductible Plan 10 contract.

In spite of non-profit operation and the lowest administrative cost in Blue Cross history, Blue Cross benefits paid to non-group $25 Deductible subscribers had continued to exceed subscriber income. The only solution for Blue Cross was to standardize the daily room and board allowance and still provide coverage in full on the vastly important hospital services.

Under the new $25 Deductible Plan 10 non-group contract the $10 daily board and room allowance will cover the large share of room and board costs in most cases.

The provisions of the new Plan 10 contract became effective with hospital admissions on and after October 1, 1954. The only exception is for maternity care. For the first nine months the new $25 Deductible Plan 10 contract is in effect, maternity benefits will be paid on the basis of the $25 Deductible semi-private contract.

All non-group subscribers were notified in writing of the change of the $25 Deductible non-group contract.

There is no change in the $25 Deductible group contract. Over 51,000 group subscribers covered by this contract will receive the full semi-private accommodation benefits in addition to coverage in full on all special services.

BOOK REVIEWS

Books listed here become the property of the Ramsey, Hennepin and St. Louis County Medical Libraries when reviewed. Members, however, are urged to write reviews of any or every recent book which may be of interest to physicians.

BOOKS RECEIVED FOR REVIEW

PROGRESS IN CLINICAL SURGERY. A Symposium edited by Rodney Smith, M.S., F.R.C.S., Surgeon, St. George's Hospital, London; Surgeon, Victoria Hospital for Children; Consultant Surgeon, Wimbledon Hospital; Late Hunterian Professor R.C.S. 414 pages. 112 illustrations. Price $7.50, cloth. Boston: Little, Brown & Company, 1954.

ANATOMY, REGIONAL AND APPLIED. R. J. Last, M.B., B.S., F.R.C.S., Professor of Applied Anatomy, Royal College of Surgeons of England. 665 Pages. 309 illustrations. Price $10.00, cloth. Boston: Little, Brown & Company, 1954.

NERVOUSNESS, INDIGESTION AND PAIN. Popular edition. Walter C. Alvarez, M.D., Emeritus Professor of Medicine, University of Minnesota (Mayo Foundation); Emeritus Consultant in the Division of Medicine, Mayo Clinic. 235 pages. New York: Harper & Brothers, 1954. Price $3.50.

NEEDED RESEARCH IN HEALTH AND MEDICAL CARE. A Bio-Social Approach. By Cecil G. Sheps, M.D., M.P.H., executive director of Beth Israel, Hospital, Boston, and lecturer on preventitive medicine, Harvard University; and, Eugene E. Taylor, M.D., M.P.H., director of communicable and chronic disease control, Louisville-Jefferson County Board of Health, Based on a Seminar in Chapel Hill, North Carolina, September, 1952. 216 pages, cloth. Chapel Hill: The University of North Carolina Press, December 11, 1954.

REVIEW OF MEDICAL MICROBIOLOGY. By Ernest Jawetz, Ph.D., M.D., professor of bacteriology and lecturer in medicine and pediatrics, University of California School of Medicine, San Francisco, California; Joseph L. Melnick, Ph.D., professor of epidemiology, Yale University School of Medicine, New Haven, Connecticut; and, Edward A. Adelberg, Ph.D., assistant professor of bacteriology, University of California, Berkeley, California. Paper cover. 360 pages. Price $4.50. Los Altos, California: Lange Medical Publications, 1954.

DISEASES OF THE SKIN. Oliver S. Ormsby, M.D., and Hamilton Montgomery, M.D., M.S. Eighth edition. 1504 pages. 750 illus. 18 colored illus. Philadelphia: Lea & Febiger, 1954.

It may be said with considerable local pride that the influence of the junior author, Dr. Hamilton Montgomery of the Mayo Clinic, becomes increasingly evident with each succeeding edition of this standard dermatologic text and reference book. Those familiar with earlier editions do not need to be told that it has been one of the great works on dermatology in this country, if not in the world. They will be pleased to see that the eighth edition is even bigger and better, showing improvement particularly in the modernization of the bibliography and in therapeutics. More and better illustrations have been added to the already outstanding earlier collection.

Somewhat of a departure has been made in the collaboration in the writing of certain sections by authorities in specialized branches of dermatology, notably DeLamater (fungus diseases), Kierland (syphilis), Farber (vascular diseases of the skin), and Lobitz (chemistry and physiology). The results of this division of labor appear to have been notably successful.

The sections on the histopathology of skin diseases deserve special mention, since Dr. Montgomery is an acknowledged expert in this field. One only wishes that the publishers could see fit to put this material in larger type, for the benefit of those general and dermatopathologists who keep "Ormsby & Montgomery" in constant use.

The new edition makes this the most modern comprehensive textbook of dermatology in the English language. It can be unreservedly recommended to anyone interested in the diagnosis and treatment of skin diseases. The reviewer has already ordered his copy.

ROBERT W. GOLTZ, M.D.

SMOKING AND CANCER: A DOCTOR'S REPORT. By Alton Ochsner, M.D. Foreword by Evarts A. Graham, M.D., New York: Julian Messner, Inc. Price $2.00.

The book begins with the uncompromising declaration, "Cigarettes cause cancer," and in his foreword Dr. Graham asks, "Isn't it time that the official guardian of the people's health, the United States Public Health Service, at least make a statement of warning"

Both Dr. Oschner and Dr. Graham are pioneers on the subject of smoking and cancer and their findings and demands are being recognized by top medical and scientific authorities.

BEYOND THE GERM THEORY. A New York Academy of Medicine Book. Iago Galdston, M.D., Editor. New York: Health Education Council, 1954. Price: $4.

As the title implies, this book broadly attempts to illustrate the emotional factors involved in health and disease states. The chapters were written by a number of authors; this causes repetition of the material (such as the history of medicine, bacteriology and nutritional diseases).

The text may be worth while to those interested in the psychosomatic problems in medicine.

DONALD S. AMATUZIO, M.D.

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